THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—12TH YEAR. SYDNEY: SATURDAY, JULY 18, 1925. No. 3.

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CANADIAN NUMBER.

CHRONIC APPENDICITIS, OR WHAT?[1]

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SOMETIMES in the whirl of a busy life we neglect other things to our own and to our neighbour's detriment. One does not vary his daily routine with a sufficient amount of recreation and hence is prone to travel in a professional rut, talking and thinking professionally, until suddenly one is jolted out of the rut and is ditched.

Because of this method of working, appendicitis as a surgical entity is a comparatively new disease, though Mestivier[1] as long ago as 1759 described a case in which he had opened an abscess near the umbilicus. The patient died and at autopsy the sloughing appendix, containing a rusty pin, was found to be the cause of the abscess.

For nearly a century isolated cases were reported, but more as a surgical curiosity than as a surgical entity. In 1848 a London physician, one Hancock,[2] successfully operated upon a case diagnosed as appendicitis.

In the 'fifties George Lewis,[3] of New York, published "A Statistical Contribution to our Knowledge of Abscess and other Diseases Consequent upon the Lodgment of Foreign Bodies in the Vermiform Appendix, with a Table of Forty Cases." Though one got little encouragement from a study[4] of the results as only three recovered, yet the paper was an "arrow" pointing the way.

In 1858 Howard,[5] a Canadian of Montreal, delivered "A Clinical Lecture on Inflammation and Perforation of the Appendix."

In 1884 Samuel Fenwick,[6] of London, published a series of clinical lectures. It was not, however, until 1886, when Reginald Fitz, of Boston, brought forth what is now considered a classic, with its clear cut reasoning, that the profession sat up and began really to take notice seriously.

In the meantime the great Lister had held up the torch of antiseptic surgery with the result that the path into the abdomen has been cleared of most of the obstacles of approach and, if opened carefully and with due regard to the patient's condition, it can be done now with safety. Even the veriest tiro enters it with impunity, though often he finds the exit strewn with the *débris* of ruined health on the part of his victim, if he escapes the death penalty, and his own already shady reputation slipping, unless perchance he is supported by a well commissioned family practitioner.

[1] Read at a meeting of the Detroit Academy of Surgery.

The first recorded death from appendicitis in the Province of Ontario was in 1897, but in the preceding ten years there was a yearly death rate of over six hundred from peritonitis and inflammation of the bowels. I merely mention this to show how comparatively recent it is that doctors have been fully alive to the pathology of the acute condition and how still more recently has chronic appendicitis been discussed.

I well remember the excitement and questionings that went on when the first gangrenous appendix was diagnosed and successfully removed in the Toronto General Hospital.

The disease as such became a recognized pathology, but the patient had of necessity to be very ill before surgeons in the early days would risk their professional reputation by removing an appendix other than one that we now speak of as fulminating.

Time passed and surgeons with the improvement in technique began to remove appendices showing a less degree of acute inflammation with great benefit to the patient and a speedy recovery.

It was observed in many such patients who had been the subject of gastric disturbance, that they were relieved of their symptoms. Later physicians and surgeons alike, when confronted by a patient with intractable hyperchlorhydria, who occasionally developed a pain in the right iliac fossa followed by an exacerbation of his gastric disturbance, naturally put it down as a case of chronic appendicitis and out came the appendix. Occasionally such patients were relieved of most of their symptoms, but as the operation became more simple and commonplace and "appendicitis clubs" began to be formed in some cities in the great American Republic, it was found that many such people were not only not relieved of their symptoms, but some seemed worse because of the operation. It then became the cue to attribute the trouble to adhesions. (What a salvation that term has been to the profession!) Some, however, disregarded the adhesions and traded the diagnosis for gall bladder disease or for gastric pathology. I am disregarding the period through which the profession passed, when the ovary was "hunted in pairs" for the relief of all the ills to which our women were prone.

Yet we surgeons for years kept taking out miserable, shrivelled-up, attenuated appendices and did not seem to be able to appreciate the fact for a long time that we were being "done" and the patient's health was not being improved by such practice.

Of course, if there is a history of a definite acute attack of appendicitis, that is a different matter and a patient's health will be greatly benefited by disposing of it as a more or less continuous focus of infection. I well remember getting great kudos from such a case. Following an acute attack he had frequent recurrences of pain, sometimes on the right side and sometimes on the left. These were looked upon as probably renal colic. When I went in to remove his appendix, I found the tip adherent to and communicating with his sigmoid. In the appendix was a large concretion which one could not expel either into the caecum or into the sigmoid. The removal of that appendix gave complete relief to his attacks of renal colic.

People do, however, have trouble in the right iliac fossa and while it is not chronic appendicitis *per se*, the appendix may be involved in a fibrosis resulting from a low grade inflammation, involving not merely the appendix, but the caecum and ascending colon as well. They come complaining of pain, lassitude, loss of "pep," drowsiness, more marked in the afternoon, loss of appetite, "indigestion" and some nausea. If the symptom of nausea is analysed, it will be found it has no relationship to the pain and is most troublesome in the early morning. These are the cases that have ileo-caecal regurgitation. Young women, in the know, often hesitate to mention this. The patient is easily upset nervously and indeed has often been passed from one to another as a confirmed neurasthenic. Usually they complain of constipation. In some cases there is mucus in the stools and occasionally blood from an ulcerated area in the caecum. One case had such a severe hæmorrhage that direct blood transfusion was required as a life saving measure.

A careful physical examination will reveal some tenderness, but no localized rigidity and under the hand one can often almost grasp a large flabby tumour. I say almost, because just as one thinks he has it, suddenly it is gone. In addition to the tenderness there is frequently hyperæsthesia, not only over the right iliac region, but also under the left costal border and there may even be tenderness over the lower left dorsal roots because of the fœtal nerve relationships. This is so different from the gall bladder case and for the reason that the caecum was originally a left-sided organ.

A barium meal may be given and the Röntgenologist will report nothing abnormal except delay in the caecum with failure to empty on the part of the appendix. If you follow the case through the series, you will be struck oftentimes with the size of the duodenum and in a certain proportion with what appears to be a "duodenal drag." Watch not only for the caecal delay, but watch also for a caecal pocket that may persist for days and watch also for the ribbon-like transverse colon. The retention in the appendix may mean anything or nothing, mostly nothing, and is by no means a reason for its removal. When this examination is completed and the bowels have been well cleared, then resort may be had to a barium enema. If there is marked atony, you will find the *haustra* ironed out and the caecum appears as a large pear-shaped mass, usually well down in the pelvis, with what may suggest a filling defect in the ascending colon due to a fan-shaped band to the outer side. Very frequently, when the enema has filled the caecum, you may be able to see the opaque material regurgitate through the ileo-caecal valve. In one of these cases the regurgitation was so marked that in twenty minutes from the time of giving

the enema we saw a well-formed duodenal cap and many times when the enema has reached the duodeno-jejunal junction, we have desisted because of the profound nausea produced. This, I think, gives a clue to the early morning nausea that is a cause of distress in many of the cases, for with the prone position and the lessening of the peristalsis during sleep, a gradual regurgitation takes place and reaches its maximum with nausea in the morning.

The "duodenal drag" or the distended duodenum that sometimes is picked up during the X-ray investigation, is caused by the lengthening of the mesentery with an accompanying tortuosity and varicosity of the superior mesenteric vessels and these in turn make pressure upon and cause narrowing of the bowel lumen where they cross the duodeno-jejunal junction. This in my opinion is responsible for the frequently associated duodenal distension.

There are many kinks and twists in the gastro-intestinal tract and most if not all of these are congenital. The kink that may occur at the duodeno-jejunal junction, is probably congenital and may never cause symptoms. The ileal kink of Lane is undoubtedly congenital. I have seen it at autopsy in a new born babe that died from hæmorrhage a few hours after birth. Then there is a triangular band of adhesions causing a kink in the ascending colon and again there is the sigmoid kink holding the sigmoid to the pelvic wall or to the left broad ligament.

The question naturally arises: if these kinks are congenital and cause no symptoms in some, why can they cause trouble in others? If you go into the history fully in these people who have had the appendix removed for chronic appendicitis and who ultimately become the bane of one's existence, passing from one doctor's office to another as confirmed neurasthenics, you will find that prior to their trouble they have met with some accident or have passed through a serious illness. The neuro-muscular control has been upset and until that has been corrected, they cannot get back to a modicum of good health.

For example and this is an extreme example, a short time ago I saw a female patient, twenty-eight years of age, who up to two years ago was looked upon as one of the healthiest girls in the community. She weighed sixty-five kilograms (one hundred and forty-three pounds). She was driving with some friends in a car when the car dropped into a culvert. She was not hurt, had no bruises and thought when she found no one was hurt, that the incident was a great joke; but she began shortly after to have "indigestion" and in a year's time was delicate and weighed fifty-six and a half kilograms (one hundred and twenty-five pounds). She was engaged to be married and the family thought she was "love sick" as the young man was not in a position to marry. The wedding was hastened and the pair were assisted to set up housekeeping. A year later I saw her. She then weighed forty-four kilograms (ninety-seven and a half pounds), quite a change from sixty-five kilograms two years ago and marriage had not been helpful. She vomited within two hours after taking food. Physical examination revealed very little except an absence of something in the right iliac fossa, and tenderness as high up as the costal margin. X-ray examination, however, revealed an atonic caecum that had failed to rotate, with the *caput* on a level with the costal margin, the appendix behind and the small bowel entering the caecum from the outer aspect. She was opened and the appendix was removed, the retaining bands divided, the caecum plicated and then fixed into a bed just above the brim of the pelvis. She had a little post-anæsthetic vomiting, but in a few days she was partaking of a full low protein diet, enjoying it and looking forward to every meal. Yet hers was a congenital condition, without symptoms for years until aggravated by an unrecognized injury to her neuro-museular control.

When these upsets come, it is well to look upon these people as having reverted to the herbivorous type of animal with the long caecum and feed them as such, largely upon a vegetable diet, getting the protein as low as is compatible with vigour, but there must be no protein in the form of meat, chicken, fish or eggs allowed. It is well also to endeavour to change the bowel flora from that of the carnivorous human to one of the herbivorous animal. To accomplish this a yeast cake eaten every night or some form of Bulgar or acidophilus bacillus may be given. Abdominal massage to improve the muscular tone may be helpful, as well as physical drill. They should also wear a corset with an abdominal lift. Some are so thin that such is not effective, when an ordinary abdominal belt with a rubber bath sponge under it will be found to give the necessary support when in the upright position. In women pregnancy will give the necessary uplift to relieve the condition and after parturition the lift may be continued by a belt. If, when these people are seen, the appendix has not already been removed, fully four-fifths may be restored to good health by such means.

If, because of a continued inability to restore the patient to good health in the manner described, it becomes necessary to resort to surgery and if this is carried out in a sane and conscientious manner, followed for two, three or more months, while a reparative process is going on, with a low protein diet, the patient will be restored to rude health in a way that will surprise the most pessimistic. When the caecum is a pelvic organ, resembling the herbivorous type and is atonic with marked ileo-caecal regurgitation, the removal of the appendix with a plication of the caecum and ascending colon and then a burying of the plicated margin into a slit seven and a half to ten centimetres (three to four inches) long in the posterior parietal peritoneum, together with a division of the sigmoid bands, will lead to a cure. How much this result may be due to a disturbance of the sympathetic

ganglia in making this bed, it is hard to say. There was no mortality in the first one hundred cases, with two deaths in the second hundred and the results so far have been striking. There has been a gain in weight of from four and a half to thirteen and a half kilograms (ten to thirty pounds), with increased efficiency and a loss of much of the neurasthenia. Some practitioners have even complained that such treatment of their patients has destroyed a weekly source of revenue!

It is advisable in all of these cases that come to surgery, to examine the state of the duodenum and if it be very atonic as well as dilated, it may be well to do a duodeno-jejunostomy at the same sitting, if the patient's condition warrants it, but, of course, this adds considerably to the gravity of the surgical procedure. In most cases it is wiser to defer such for a second operation, when the patient has derived what benefit he can from the plication and fixation, when the resulting daily evacuation or more has become a habit, thus adding to the joy of living.

In the advanced cases where there is marked atony with third degree incompetency of the valve and deep pigmentation of the caecum and ascending colon, with a history of long continued ill health, where the appendix has been removed and a subsequent series of adhesion-breaking, health-breaking and heart-breaking operations have already been done, nothing short of a radical resection of the terminal ileum, caecum and ascending colon at a point above the pigmentation, with an end-to-end anastomosis of the cut ileum to the ascending colon or to the hepatic flexure as the case may require, will be of any benefit. The extraordinary thing is that Nature has some means whereby a partially reconstructed valve developes at the site of anastomosis.

Some years ago we abandoned the side-to-side anastomosis because three cases returned for treatment that had developed pockets in the blind ends, but in adopting the end-to-end anastomosis we ran into occasional fæcal fistulæ and a higher mortality. In the autumn of 1920 we adopted the use of a piece of rubber tubing as a temporary connexion at the site of the anastomosis and since then we have had many cases with one death and this was in no way attributable to the operation and there have been no fæcal fistulæ.

The operation of resection has been carefully worked out. First the mesentery is perforated at the site of the proposed section of the colon. The same is done at the ileum. Then through this perforation in the mesentery beginning at the ileal end a double clamp (Ochsner forceps) is applied to the mesentery and this is cut between; another two pairs of forceps are applied and the mesentery cut. This procedure is continued until the hole in the mesentery at the site for dividing the colon is reached. The colon and the ileum are then cut across by means of a cautery knife and the portion of bowel disposed of. One now has a clean area. A double No. 2 iodine or plain catgut suture threaded on a semi-curved round needle is passed around the points of the last pair of forceps applied to the mesentery at the colic end and this suture overcasts the forceps with several loops until it overlaps the points of the next pair. The forceps are then loosened and withdrawn, while the suture is tightened. This procedure is continued until the entire cut edge of the mesentery is oversewn and all bleeding thus controlled. The two ends of the suture are then tied and left long, when one finds the free ends of the bowel to be anastomosed lying side by side. The junction is made in the usual way, using the Balfour idea for enlarging the lumen of the ileum. When the inner suture of chromic gut has reached the point opposite the mesentery, a piece of rubber tubing with a 12.5 millimetre (half inch) lumen and about 37.5 millimetres (one and a half inches) long is caught by the suture and the remaining part of the closure is completed over the tube. The serous suture is then completed and the ends of the serous suture and the mesenteric suture are then tied, thus closing off any possible opening between. The rubber tube maintains the lumen of the gut during the temporary œdema that occurs while healing. It is passed *per rectum* in from five to seventeen days. This is usually preceded by a few colicky pains for a day or two.

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(3) George Leavis: "A Statistical Contribution to Our Knowledge of Abscess and Other Diseases Consequent upon the Lodgement of Foreign Bodies in the Vermiform Appendix," *The Medical Record* (New York) ————, 1856, page ——.

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(5) Howard: *Montreal Medical Chronicle*, 1858.

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ACUTE SEPTIC OSTEO-MYELITIS.

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The diagnosis of acute septic osteo-myelitis is rarely made in the first week of the disease. This is an extraordinary fact, in view of the frequency with which the disease is met and the constancy of its symptoms. It is my intention to outline some of the salient points concerning osteo-myelitis that have been brought to the attention of the staff of the Hospital for Sick Children. These points have been noted from a study of the histories of the cases past and present.

Ætiology.

This disease is to be found in its primary attack only up to the age at which the last epiphysis unites. It is a disease intimately connected with epiphyseal lines. It never occurs after fusion of

the epiphysis. I shall discuss this point more fully farther on in this paper.

Infection.

The infection is always blood borne and the organism is generally the streptococcus or staphylococcus in one of its forms. A bacteriæmia or a septicæmia must exist. If the organism is a streptococcus, entry into the blood may occur *viâ* the tonsils and throat. This organism is not the one most commonly found causing osteo-myelitis. The great majority of cases are due to some form of the staphylococcus.

The entry of the staphylococcus to the blood stream seems to be in practically every case from a superficial skin lesion. Since the association of a skin lesion was first noted in our cases, every subsequent case has demonstrated it. The skin lesion may be a furunculosis and when one sees the frequency with which furuncles exist in the presence of bone lesions, one must realize that a bacteriæmia is a not uncommon occurrence in furunculosis. Again, the skin lesion may be an infected wound or abrasion, one that may have a scale covering it under which a drop or two of pus may be found. One of these two types of skin lesion is the invariable accompaniment of staphylococcal osteo-myelitis. Blood cultures taken during the first few days of the disease demonstrate the presence of organisms.

Injury.

Before a local condition developes in the presence of a bacteriæmia, there is most likely a point of lowered resistance and the localization of the lesion in this disease is determined by the point of lowered resistance. This lowered resistance point seems to be at the site of an injury. The history will show us that the site of the disease was formerly the site of a "sprain." This sprain was sufficiently severe to cause a limitation of activity of the limb and when movement or work was attempted, pain was caused. The pain was not sufficiently severe to cause the patient to lie up, but it was sufficiently severe to cause a limp, if the sprain was in the lower limb. The duration of the symptoms of this sprain was short; it usually cleared up in a day or two and was promptly forgotten until the trend of subsequent events recalled it to the patient's mind.

The pathology of these sprains seems to be that an injury is produced to the end of the diaphysis at the epiphyseal line. It would seem that in a person who has a cartilaginous epiphyseal line, an extra pull on a joint ligament or capsule would not rupture the capsule or ligament, but would pull the epiphysis to such an extent that damage would result at the junction of the epiphysis and the diaphysis. Hæmorrhage would result and in this small hæmatoma an area of lowered resistance would exist that would be a favourable location for an infection. That this theory of injury at this particular region is borne out clinically is evidenced by the site of the infections in certain locations. When the lower end of the femur is involved, it is always the *planum popliteum* that is found denuded of periosteum. This is because the injury causing this localization has been a hyperextension of the knee. When the upper end of the tibia is involved, the lesion is close to the tubercle. The tubercle is the attachment of the patellar tendon and as such liable to great strain. Moreover, it is liable to direct injury.

Pathology of Lesion.

The infection of the disease always localizes at the point of strain and this is to be looked for and found at the diaphyseal side of the epiphyseal line. Beginning here as a small area of infection, the inflammation spreads equally in all directions and it soon reaches the periphery of the bone, where it comes to involve the periosteum. The involvement of the periosteum is such that pus forms under it and it is raised from the surface of the bone. In this process the blood vessels entering the bone from the periosteum are torn out of the bone and to them adhere some osteoblasts which survive to form the involucrum.

This stripping of the periosteum continues until drainage is established. In addition to this rapid spread of infection under the periosteum there is a a corresponding spread into the depths of the bone, so that within a week of the onset of the disease the whole diaphysis may be involved, with pus completely surrounding it and its medulla full of pus. This stage is the picture of the disease given in most text-books on surgery. The entire segment of the limb is swollen and the condition looks like a massive cellulitis. This stage bears in reality the same relation to acute septic osteo-myelitis that secondary general peritonitis bears to acute appendicitis.

The infection in the bone always begins in the diaphysis close to the epiphysis. We have seen no early cases where pus was demonstrated in the medullary cavity, although we have on occasion deliberately opened into the middle of the shaft which appeared normal, and taken smears from the medulla. After closing this wound, we have then attacked the end of the diaphysis where it was obviously involved. In later cases, those that have existed a week or ten days from the first pain, pus is found in the medulla. This is most probably due to the advance of an unmolested infection. We believe that the advance of the disease locally may be checked in certain cases by a prompt interference in the first hours of the disease. It is possible by prompt action in draining the local lesion to prevent the localization from becoming a septicæmia.

Types of the Disease.

There are two main types. The first one is that in which the bacteriæmia, giving no symptoms, at once changes into a septicæmia giving an intense general reaction. In a short time, one or two days, it may have developed as an incident in its course a bone lesion. The development of this bone lesion and the prompt treatment of it seem to have no

effect on the general infection. This type of case is for the most part fatal.

The second type is a different story and is the disease with which we are here concerned. The bacteriæmia is symptomless and the first thing of which the patient complains is pain.

Symptoms.

Pain is the first symptom noted and at first is very slight, but it rapidly developes to be a pain that demands attention. It is progressive and has no remissions. Soon after its onset, within twenty-four hours, there is obvious general reaction, fever, increase in pulse rate, loss of appetite and nausea. On examination the limb involved is beginning to be held in a guarded position. The patient says the pain is at the joint. Inspection shows a limb and joint normal in contour; no redness or swelling is discernible. On careful palpation around the joint, tenderness may be elicited and the tender spot will be found to be small in extent and to lie exactly over an epiphyseal line. It is to be noted that this tender spot is at the epiphyseal and never at the middle of the shaft. As the disease advances, the pain becomes increasingly more severe; sleep becomes impossible; the limb is held partly flexed and rigid; the patient complains bitterly of any movement of the bed or his person. After eighteen or twenty-four hours there is severe general reaction and agonizing pain, said to be at the joint; tenderness increasing, but yet no swelling nor redness; the limb appears normal. The case is at this stage generally diagnosed as rheumatic fever. After twenty-four hours swelling may occur over the spot of tenderness, but swelling may not occur until as late as forty-eight hours. When it does occur, it means that the infection is involving the periosteum and the superficial tissues. It will be found in all cases that the swelling begins close to the joint and extends and progresses away from the joint. Once the swelling begins, it increases rapidly and the general symptoms abate somewhat. After five or six days the pain becomes much less severe; sleep is possible, but the limb is held immobile. It is very much swollen; the whole length of the bone is now involved sub-periostally and the medulla also. A fact that is of the greatest importance in the formation of a diagnosis of rheumatism in these cases, is that very frequently a second lesion appears within a few days of the first. This seems to be contributory evidence of a polyarthritic condition—rheumatism.

Diagnosis.

The diagnosis is made on the following points:

Age.—Only during the existence of unfused epiphyses.

Injury.—History of a sprain to a joint.

Skin lesion.—Furuncle or infected abrasion or cut.

Pain near a joint, unremitting and progressive.

Tenderness at site of pain; this always over an epiphyseal line.

Inspection during first twenty-four hours normal.

General reaction and positive blood culture.

Treatment.

Treatment must be prompt and we believe that early treatment of the lesion may be a great factor in preventing the bacteriæmia from becoming a septicæmia. Again an early drainage certainly tends to limit the spread of the infection in the bone. In this way the shaft of the bone is not lost.

When a case gives the symptoms suggestive of an osteo-myelitis, it is wise to proceed at once to open the integuments at the site of tenderness. The periosteum is cut down upon and if one is sufficiently early in the disease, a periosteum with œdema is the only abnormality in the soft tissues. On division of this the bone underlying seems normal. This should not deter one from making an opening in the bone down towards the epiphyseal line. One will here find a small collection of pus or infected bone. A small window should be cut to permit of adequate drainage and the release of the pressure of infected inflamed tissues. In cases where infection is a streptococcus, a thinnish pus may be found and it is our experience that this type of case heals promptly without the formation of a sequestrum and the bone destruction common to the staphylococcal variety of infection.

In an advanced case the difficulty will be to differentiate it from a cellulitis. In this case the infection may have spread through the capsule of the joint and an arthritis be present. Whenever this occurs, the joint must be drained promptly.

Unfortunately radiographs are of no assistance in the diagnosis of an early case. The first positive information given by these films is at about three weeks, when one can see the beginning formation of the *involucrum*.

CAUSES OF CHRONIC PAIN IN THE BACK AND BUTTOCKS.[1]

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One of the commonest and most troublesome complaints with which medical practitioners have to deal, is chronic pain in the back. It occurs in many different types of patients. It is common in healthy people and debilitated people, in men and in women. It is rare in children. In this paper it is proposed to deal especially with the diagnosis of the cause, although it must be admitted that treatment is not always easy even when the cause is known. The cases are divided into four main classes:

1. Those due to local causes, that is to disease of the structures of the back, such as nerves, muscles, bones and joints. These are the "sore" or "rheumatic" backs.

[1] A lecture delivered to several county medical societies under the Ontario Medical Association scheme of post-graduate education.

2. Those due to gross lesions of the thoracic, abdominal or pelvic organs.

3. Those where pain in the back is the result of constitutional conditions causing general debility, such as anæmia and faulty nutrition. In this class should be included most of the cases of neurasthenia who complain of back pain. These are well described as "tired" backs.

4. Those cases where pain in the back is a hysterical or protective phenomenon. These patients have often started with a true pain in the back, but later the symptom is used to protect them in a dilemma.

Pain in the Back Due to Local Causes.

Nerve Lesions.

Injury or disease of nerves are rare causes of pain in the back. When a nerve is pressed upon by a bone or a tumour, the pain is severe and agonizing and the distribution of the pain is limited to the area supplied by one nerve or spinal segment. This is seen occasionally in tumours and tuberculosis of vertebræ, but otherwise almost never. *Herpes zoster* in elderly people may leave a painful and tender area for months or years. The diagnosis of the cause is made from the history, the scars and the area of hyperæsthesia. The treatment is difficult.

Disease of Muscle.

In this class are included so-called myalgia, torticollis, intercostal neuralgia, lumbago, sciatica and "rheumatism." The large majority of chronic sore and stiff backs in army, out-patient and private practice has convinced me that muscular rheumatism as a separate disease does not exist. Further, I am convinced that the pain of lumbago is not felt in the muscles or fibrous muscle sheaths and that fibrositis, except as it may occur as the early symptom of the condition which later developes into osteo-arthritis, is not a cause of pain in the back.

Careful examination of several hundred cases of lumbago has revealed the absence of any detectable tenderness in the lumbar muscles with the exception of a few cases where tenderness was doubtful, but could not be excluded. In confirmation of this is the fact that myalgia and fibrositis, apart from the pains of "stiff muscles" the result of unusual exercise and osteo-arthritis, cannot be found elsewhere in the body and it is probable that lumbago would never have been described as a muscular disease were it not that vertebral joints are so deeply buried by overlying muscles that their examination by palpation is impossible. Inflammation of muscles as a cause of pain in the back, I believe, can be disregarded. Tired overworked muscles may be the cause of tired backs, but they are not the seat of pain in sore backs or lumbago. After unusual exercise the back muscles may be stiff and sore, as the muscles of the arm are sore after unusual exercise such as digging, pitching hay and so forth. This form of pain in the back is a temporary one and where pain persists after several days, another cause should be sought.

Fibrositis and Osteo-Arthritis.

Osteo-arthritis of the vertebral joints is very common. It is infrequent before the thirty-fifth year and is increasingly more frequent after that as age advances. Its onset is seldom sudden, but relief may not be sought until there is an acute attack. The condition is often complicated by an injury such as a sprain, fall or a heavy lift. These acute attacks of lumbago may occur at long intervals with no pain between attacks. They are precipitated by exposure to wet and cold, but are most frequent in my experience during the season when acid fruits, especially strawberries, tomatoes and grapes, are freely eaten. Other patients without acute attacks may complain of a stiff, slightly painful or aching back which may be stiff on arising in the morning, but "limbers up" as the exercise of the day is carried out and the day becomes warmer. In some cases the pain is most troublesome after the day's work is over. If a careful history is obtained, it will usually be found that there is occasional pain or stiffness in other joints. A frequent history is that for a few days there are stiff fingers or an aching shoulder, then stiffness in the neck or slight occipital headache in the early morning followed a few days later by slight thoracic pains, as the inflammation leaves the cervical and descends to the thoracic joints and often ends with a few days of mild lumbago or sciatica. A frequent association of osteo-arthritis is so-called acid indigestion. On physical examination commencing Heberden's nodes or crepitus in shoulders or knees or other joints is usually found. Examination of the back will reveal in all but the slightest cases some restriction of movement in the back. In all cases of pain in the back every joint in the body should be examined.

A study of the early stages of osteo-arthritis shows that often for years before a joint proper is affected, pain may be present and tenderness. This tenderness is usually localized to a small area where a muscle tendon or ligament is inserted about the joint. As the disease progresses, more and larger areas are affected and finally the joint becomes involved. The use of X ray photographs has shown very clearly that after attacks of lumbago become more severe and of longer standing, osteo-arthritic changes occur at the attachment of the back muscles to the vertebræ. The early inflamed tender spots at the point of attachment of the muscles explain why the lumbago patient has most of his pain when he contracts his back muscles as in rising from the stooping position and yet has no pain when his vertebral joints are moved by passive movements.

Rheumatoid and Infective Arthritis.

Rheumatoid and infective arthritis are usually more acute and occur more frequently in younger people. The frequency of a gonorrhœal history is significant, although the gonorrhœal infection may have preceded the pain in the back by many years. In these cases a urethral smear following prostatic massage often reveals the presence of a latent prostatic infection. A history of arthritis in other joints, more acute than an osteo-arthritis, is fairly

common. The onset is usually more acute and pain is greater than in the chronic variety of osteo-arthritis and loss of weight and strength may occur. Slight fever is usually present.

On examination rigidity of the back is more marked. In some cases the rigid "poker back" is found. The attack subsides more slowly and ankylosis may occur. The vertebral joints from occiput to sacrum may all be affected at once.

Method of Examination of Back.

A proper clinical method of examination is of the highest importance in diagnosis. The large amount of muscle and fat tissue overlying the vertebral joints makes palpation of little use. The patient should be stripped down to the level of the great trochanter and stood with the back to the light and the examiner should sit squarely behind him. After inspection the muscles of the back should be palpated to see if there is any tenderness. This should be done by pressing the thumbs deeply into the lumbar muscles. Care should be taken to make the patient distinguish between tenderness in an area and the pain which is felt in the same area. Malingerers always exaggerate the tenderness of pressure. After testing the tenderness to deep pressure, the patient should be made to bend far forward with the chin touching the chest to see the shape of the back bow. In all but obese and elderly patients this should be an arc of a circle. An area of restricted bowing should be noted. It may be wise to turn the patient sideways to view the bow of the back. In very slight and early cases of osteo-arthritis there may be no restriction of movement. In all other cases of osteo-arthritis, in rheumatoid arthritis and in caries of the spine movement is restricted to some extent. The next test is to make the patient bend backward to the fullest extent with the chin elevated, while the examiner's thumbs are pressed deeply into the lumbar muscles on both sides. In fibrositis and osteo-arthritis most pain is complained of when the patient is returning to the erect position. The area of restricted movement may still be present. In the case of the malingerer or the patient exaggerating his pain who complained of tenderness on deep pressure before, no complaint of pain is made when the weight rests against the thumbs pressed deeply into the back. The patient is too much engaged in keeping his balance to remember to complain of tenderness. Having examined the forward and the backward movements, the movements and shape of the side bow of the back should be tested by getting the patient to bend to the right and left with the under ear pressed over the corresponding shoulder. The area of restricted movement is again demonstrated. Restriction of movement in the thoracic area is harder to judge owing to the ribs restricting all movements of the thoracic vertebræ.

The patient should then sit on an examining table and bend forward. If he has purposely restricted his movements in the pervious tests, the bow may be found to be perfect in this movement. Then lay the patient on his back with the thighs and legs flexed. The examiner stands on the patient's right with his back to the patient's head, places his left arm under the patient's flexed knees and rolls him up on to his shoulders. The examiner looking down the crease of the back, observes the bow again. This manipulation may be necessary because in disease of the sacro-iliac or hip joints forward bending may be restricted, but when the thighs are flexed as in this manipulation the bend of the back is not restricted by disease in the hip and sacro-iliac joints.

Further test of the movements at the vertebral joints should be made by having the patient lie prone with the arms at the sides. The examiner facing the patient places his right arm under the thighs just above the patellæ and raises the thighs until only the chest of the patient rests on the table. The back bend and the side movements may be thoroughly tested in this way, but mild cases of osteo-arthritis, who showed restricted forward bend, may show no limitation of movement by this test. In more severe cases of osteo-arthritis, in rheumatoid arthritis and in caries of the spine restricted movement will always be apparent. If movement is normal as shown by the above tests and if there is no sign of arthritis in the other joints of the body, arthritis and caries of the spine can be excluded in nearly all cases as the cause of pain.

In considering the vertebral joints it should be borne in mind that there are joints joining the articular processes as well as the joints forming vertebral bodies. These articular process joints are close fitting and become painful even if slightly diseased and more movement takes place at these joints than in the joints between vertebral bodies because they are farther from the pivot point than the vertebral body joints. The latter do not fit closely, so that there may be considerable osteo-arthritic changes around them without causing any inconvenience. On the other hand a small amount of inflammation or osteo-arthritis in the joints between articular processes may give rise to much pain and disability. The most painful and crippling arthritis is found when X rays demonstrate changes in the joints between articular processes.

Sciatica and Sacro-Iliac Disorders.

Sciatica is the most serious of the so-called neuralgias. In text-books it is described as a neuralgia of the sciatic nerve which sometimes becomes a neuritis of the same nerve. It is remarkable that no demonstrable pathological condition of the nerves is found at *post mortem* examination of these cases. A few text-books have recently drawn attention to the fact that it is difficult to differentiate some of these sciaticas from sacro-iliac disease. The facts of the matter are that about 95% of so-called sciaticas are fibrositis if around the sacrum or are due to arthritis of the sacro-iliac joint or lumbo-sacral articular processes joints. Sometimes there is a subsequent neuritis of the sciatic nerve. The

other 5% include pelvic and intra-pelvic tumours, tuberculosis of the sacro-iliac joint, rare disorders such as sacro-iliac sprains and slips and sacralization of a transverse process of the fifth lumbar vertebra.

A consideration of the anatomy of the region is necessary. The sacrum is wedged in between the ilia and joined to them by strong surrounding ligaments and fine intra-articular ligaments. The joint surfaces are in very close apposition and the joints contain a small amount of synovial fluid. There is normally a slight amount of movement in the joints. The sciatic nerves lie over the front of the joints, being separated from them by the ligaments only. The transverse processes of the fifth lumbar vertebræ are usually roughly quadrilateral in shape and broader than the transverse processes of the other lumbar vertebræ. Not infrequently the processes instead of tapering toward the ends are bulbous on the lower border and may even articulate with the ilium or sacrum or both. These articulations may be the seat of arthritis.

When inflammation occurs in sacro-iliac joints, pain is referred to the buttocks and upper third of the back of the thigh, but in severe cases the pain is referred as low as the heel. This pain is not due to neuritis, but is in the referred pain area of the sacro-iliac joint and is similar to the referred pain area of the hip joint, which is down the front of the thigh to the knee and to the area of referred pain of the shoulder joint which is down the upper half of the arm. Such is the nature of the pain which is commonly called sciatica. In most cases the sciatic nerve is not affected and there is no tenderness on pressure over the nerve as it emerges from the great sciatic foramen, but in a few cases the nerve itself is affected and there is tenderness along the course of the nerve with marked atrophy and weakness of the muscles supplied by it. Milder degrees of atrophy of the thigh and calf muscles may occur without neuritis of the nerve, just as there may be atrophy of the muscles of the arm following arthritis of the shoulder joint or atrophy of the thigh muscles following arthritis of the hip. Some cases of arthritis of the shoulder joint may be followed by neuritis of the ulnar nerve. That lumbago and sciatica often occur together is to be expected, for arthritis of the lumbar and sacro-iliac joints and fibrositis around these joints frequently occur in the same patient.

The importance of recognizing sciatica as due to trouble in the sacro-iliac or lumbo-sacral joints is that treatment directed to the sciatic nerve brings no results, while counter irritation over the affected joint and orthopædic appliances are of the greatest aid in treatment. These treatments are useful allies to the general treatment of the arthritis which is of primary importance.

Treatment of Arthritis of the Spinal Joints.

Since arthritis of the spine is of two main varieties, first osteo-arthritis and second rheumatoid or toxic or infective arthritis and as the treatment of these diseases is so different, it is wise to discuss separately the treatment of each variety.

Fibrositis and Osteo-arthritis.

The ætiology of this disease is far from clear. Osteo-arthritis and other forms of arthritis being so frequently found in the same individual has made the study difficult. It is a general belief that rheumatoid arthritis is probably of infective origin, that is that it arises from foci of infection, teeth, urethra and tonsils frequently and less frequently from sinuses, gall bladder and septic sores or wounds. That foci of infection play any part in the ætiology of osteo-arthritis is doubtful. Its onset is most commonly at the age when exercise is diminishing and yet intake of food has not diminished, while the excretory organs may begin to be less efficient. In its acute phases in Canada it occurs most commonly during the months of June, July, August and September when the consumption of acid fruits is greatest. Attacks may be precipitated by exposure to wet and cold. The aching back is most troublesome before rain and after a long cold drive or after sitting in the cold after being overheated. It is apparently more common in people who have a dry skin and do not perspire freely. Pemberton, Almon Fletcher and others believe that all arthritis is more common in people showing a poor tolerance to sugar, that is in people whose blood sugar rises to an unusual degree after ingesting one hundred grammes of glucose, taken after a fifteen-hour fast, or whose blood sugar level is slower in returning to normal after taking the glucose meal. Certainly a large proportion of osteo-arthritic patients take a high carbohydrate diet. In my experience it is as common in private as in hospital practice. An interesting association is the relationship between pyloric spasm or gastric hyperacidity and osteo-arthritis. About 50% of my private cases of osteo-arthritis complained of attacks of "hyperacidity" as well. In these cases the gastric and rheumatic attacks frequently occurred together. Many patients who have suffered from osteo-arthritis ultimately die of chronic Bright's disease. Constipation aggravates the disease. Most acute attacks of osteo-arthritis and lumbago will disappear in a few days without treatment. Therefore it is very easy to be misled as to the results of treatment. As a result the laity have many cures for rheumatism. One Irishman insisted that blistering was the best cure for rheumatism because his father had been badly blistered when the liniment which he had rubbed on his rheumatic knee, caught fire and the rheumatism promptly disappeared. He almost insisted on trying this treatment when his son had rheumatic fever. It is certain that osteo-arthritis is usually aggravated by eating freely of strawberries, tomatoes and grapes or by drinking acid wines. The main indications for treatment are to study the whole condition of the patient, his exercise, his diet, the condition of his skin, kidneys and bowels. Where faults are found, endeavour to correct them. If the patient is over-eating, decrease his diet, but especially his carbohydrates and all acid fruits and

wines should be forbidden during an attack and only taken in moderation afterwards. Carbohydrates should be sharply restricted during an attack and never allowed in excess in the future.

During an attack Turkish baths or cabinet baths should be given every second day and water drunk freely. Between attacks it is well to keep the sweat glands active by warm woollen or silk underclothes and fluids should be drunk freely. In some cases six glasses of water daily will keep the patient free from attacks. The bowels should not be allowed to become constipated. Exercise should be taken regularly. Complete rest should be allowed only during the acute stages. For the pain local applications of rubefacient liniments, hot applications, cupping and mustard plasters are useful. In my experience diathermy and radiant heat have been the most useful of all treatments for relieving pain. Salicylates with alkalis should be given freely during acute attacks, but they should never be relied upon to cure the disease. They are palliatives only. Codeine or morphine may be used during the acute attacks. I have had no experience with puncture of the lumbar muscles.

Where dental root abscesses or definite foci of infection are found, it is unwise to leave them alone, but it is not justifiable to remove teeth on suspicion and I have never seen any improvement which I felt satisfied was due to tonsillectomy.

Rheumatoid Arthritis.

The treatment of this disease is entirely different. Focal infection is the main cause to be looked for, but to eradicate the focus of infection is not the whole treatment of the disease. The original focus of infection may have subsided or been cleared out and still the disease progresses. The infection has long since spread from the original focus and the joints are infected. Therefore one has three important points to bear in mind: (i.) Clear out the original focus of infection; (ii.) treat the infection in the joints; (iii.) raise the patient's resistance to infection. In looking for the focus of infection it is well to trace the history back to the onset of the disease. Was there any infection just previous to the onset of arthritis? It is not uncommon to see tonsils and teeth removed when the history is that the patient had septic finger or gonorrhœa immediately preceding the onset of arthritis. When rheumatoid arthritis followed a sore throat, it is wise to remove the tonsils. The prostate and seminal vesicles should be examined. Treatment by vaccines is worth trying, but the results are disappointing. A common mistake is to overlook the general health of the patient. If the appetite is poor or the patient emaciated, forced feeding of milk and eggs often gives excellent results. The benefit of fresh air, sunlight and cheerful surroundings should not be forgotten. Rest with the back in the straight position is essential. These patients should spend many hours each day lying flat on the back without a pillow. The joints should never be allowed to ankylose with the back in a markedly curved position.

Disease of Bones.

Diseases of bones without involvement of joints is not a common cause of back pain. Rickets, infantile or adolescent, may lead to kyphosis or scoliosis or a combined deformity. These deformities are never painful, but such backs tire easily and may be more properly considered under the type of tired backs. Tuberculosis of the spine or Pott's disease, occurring most commonly in children, should be easily recognized if the patient is examined as described above. To make a diagnosis between tuberculous and non-tuberculous caries usually requires an X ray examination. Tumours of the spine are not common and diagnosis then is only made certain by X ray examination. In such cases the nerves may be involved.

Pain in the Back Due to Thoracic, Abdominal and Pelvic Disease.

When local disease in the back has been excluded by the above examination, attention should then be paid to the organs of the thorax, abdomen or pelvis to see if they are the seat of organic disease. In the thorax mediastinal tumours and aneurysm are the only common causes of pain which may be referred to the back. Clinical examination or X ray examination will usually reveal them if present. The pain in such cases is usually in the region of the upper dorsal spines and never is referred to the lumbar or cervical areas.

Disease of the kidney is the most common abdominal cause of pain in the back. Acute nephritis may cause pain in the back, but chronic nephritis never. It is humiliating to see cases of chronic pain in the back treated by medical practitioners for chronic Bright's disease, because of the common erroneous idea of the laity that pain in the back is usually due to the kidneys. Renal abscess, tuberculous or otherwise, may cause a chronic ache or pain in the back, but the pain is unilateral and microscopic examination of the urine makes the diagnosis clear. A more common cause of slight backache is chronic or subacute pyelitis. It is common at all ages, very common in pregnant women and is usually due to the colon bacillus. Pyelitis is frequently overlooked, as it may not be revealed by chemical examination of the urine, but microscopic examination shows pus cells and motile bacilli.

Prostatitis in my experience does not often cause pain in the back nor does enlarged prostate, malignant or otherwise, unless metastases have resulted. On the other hand malignant disease of the prostate as a possible cause of sciatica should never be overlooked.

When considering uterine disease or misplacements as a cause of chronic back pain one is on very debatable ground. My experience makes me take a strong position against uterine displacements, retroversions and cervical lacerations as a cause of chronic back pain. It is true they are often found in association with pain in the back, but replacement by operation or pessaries or repair of the lacerated cervix seldom or never result in cure of

the pain. It is true that tired, debilitated women are subject to pain in the back and atony of the uterine ligaments. Rest in bed for a few weeks as after an operation always ameliorates the pain of tired, debilitated women, but the pain returns soon after they resume their work. The temporary relief of pain in these cases is to be ascribed to the rest which followed their operation. Uterine prolapse which results from severe tears of the perineum, may in some cases cause back pain, but I believe it wise to attend first to the general health of the patient and to resort to operative procedure only when these measures have failed. Organic uterine and ovarian disease, such as tumours and abscesses, are to be estimated differently for they may cause pain in the back. On the whole, uterine disorders, cervical and perineal tears have been given far too much consideration in judging the cause of chronic lumbar pain.

Constitutional Disorders as a Cause of Back Pain.

This constitutes by far the largest group of painful backs in women. Women as a rule eat too little or work too hard for the amount of nourishment they take. They also worry more than men. Women are especially inclined to shun protein foods, as meat, eggs and milk. Their preference is for vegetable salads, tea and coffee. Women who eat freely of proteins, are seldom ill and seldom tired. The "chronic neurasthenic" woman usually has a history of nervous or spastic indigestion and gradual reduction of diet because she is afraid that various foods will give her indigestion. This has been followed by loss of weight and energy, chronic constipation and then chronic debility, visceroptosis, pain in the back and flatulence. If added to faulty diet a woman works too hard and has much worry, chronic neurasthenia with tired aching back is almost sure to follow. This class constitutes the great majority of the tired aching backs in women. The "tired" back is much more common in women than the stiff and sore arthritic back.

Other causes beside lack of food which cause debility in women are slight hyperthyreoidism, chronic constipation and menorrhagia. A thorough investigation will usually reveal the cause of the debility and with the cure of the debility the pain in the back disappears.

A very striking case of this type was seen in a woman of twenty-one years who complained of pain in her back which had been so severe that for seven years she had been unable to do any work except the lightest housework such as drying dishes and dusting. Her back was always tired and life was burdensome. She had had massage, spinal jackets and much medicine all without relief. Physical examination was quite negative. Her diet was entirely lacking in fresh food and almost no fruit or green vegetables had been eaten for years. Only salt meat had been taken. She was also very constipated. Given a diet of fresh food and bran, her constipation was quickly relieved. In two weeks the pain in her back was much diminished and she felt much stronger. In three months her back was not troubling her at all and she was able to do heavy housework without trouble. This case is cited as an example of the advantage of a minute investigation of the diet and general functions of the body.

Many cases of tired back cured by rest and forced feeding of milk and eggs could be cited. The pain in the back disappears as weight and strength increase.

Pain in the Back as a Protective Symptom.

When history and physical examination have revealed no cause for pain in the back, one must always bear in mind the possibility of the pain complained of being the result of functional nervous disorder. Such cases are not rare especially in military or factory practice and where the question of compensation is involved. Apart from these there is a smaller class, more common in women, where the pain is "hysterical" or used as a protective measure to aid in an emotional struggle.

The malingerer or the man who exaggerates his pain, can usually be detected by the facts that (i.) the pain described does not correspond to the type of pain caused by arthritis, injury, thoracic, abdominal or pelvic disease and the patient appears to be healthy, (ii.) the pain is usually described as severe and all symptoms are exaggerated and (iii.) tenderness on pressure over the spine apparently causes pain when the patient realizes that the pressure is being put on, but if he or she is made to bend far backward and heavy pressure is applied, no complaint is made, the patient's mind being occupied with maintaining the balance.

Where the complaint of pain is regarded as a protective phenomenon great care and tact are required in inquiring into the emotional state of the patient. Examination having been "negative," it may be well to start the inquiry by such remarks as: "I have examined you thoroughly; I can find no cause for pain in your back. I suspect you have lost confidence in your back" or: "I suspect that your nerves are out of order. Have you any cause for worry or have your nerves been upset?" Such remarks may be answered by the patient admitting that he or she is very worried or the answer may be of such a kind that the examiner is made suspicious that such is the case. Two cases may be quoted as examples.

Mrs. K. had been a very healthy woman until she wrenched her back playing golf. After this she was in bed for eight months and received many varieties of treatment without any benefit. She was referred to me by an orthopædic surgeon who suspected an emotional disturbance. Her physical examination was entirely "negative." Careful questioning revealed the fact that she was much upset over an unfortunate love affair and her painful back was used as a protection to prevent the resumption of marital relations with her husband. She was greatly helped by being able to discuss her dilemma which she had never been able to do before, and soon came to the conclusion that she must get her emotions in hand and resume her normal married

life. She was allowed a month in which to recover her control and given a special diet to serve as a reason for her recovery. In this way she was able to explain to her husband and friends her rapid recovery. At the end of a month there was no complaint of pain or disability in the back and she has remained "cured" for many years.

The second case illustrates how a painful back may be protective in another way. The healthy wife of a divinity student complained of severe pains in her back of sudden onset. The pains only occurred on awaking, lasted for an hour each day, then disappeared and did not prevent her from doing her housework. Such pains, of course, do not correspond with the pains of any disease. The examination being "negative," she was so informed and asked about her worries. She finally admitted she had been much worried because her husband had volunteered for the foreign mission field and while she was "anxious for him to do the Lord's work wherever it was necessary," she admitted that she and her family were much opposed to him going as a missionary, because he was considered unsuitable for the work and she thought he was much better in parish work at home. She insisted that she was quite willing to go to the foreign field with him, but admitted that, when she was examined to see if she was physically fit so that she might accompany her husband, she was very disappointed when she was pronounced sound. Their tickets were bought when she suddenly developed pain in the back and was put to bed. As a result their trip was cancelled and another missionary sent in place of her husband. Her husband was informed as to the state of affairs, withdrew his request to be sent to the foreign field and the pain in the back disappeared promptly. To save her reputation in her community massage was ordered for a few weeks and she was thus able to account for her cure to her friends. This was necessary to prevent gossip.

Such cases are not very rare, but it may be difficult to get to the bottom of the emotional upset. Where the pain in the back is used as a protective measure, it is always necessary to find a way out, so that the cause of the trouble will not be known to others. Unless an excuse is found and the problem solved, the protective need is still present and a cure is difficult.

SUMMARY.

As a result of a careful clinical study of painful backs, lumbago and sciatica extending over a period of ten years I believe the following statements are justified.

1. Chronic torticollis, intercostal neuralgia and lumbago are due to fibrositis at the attachment of muscles to vertebræ which is the early stage of osteo-arthritis of the vertebræ.

2. Sciatica in most cases is due to fibrositis about the sacrum or arthritis of a sacro-iliac or lumbo-sacral joint, but may be complicated by neuritis of the sciatic nerve.

3. In women the commonest form of back pain is the tired back which is usually associated with under-nutrition. Lack of protein in the diet is the most common cause of these tired backs and super-alimentation with milk, eggs and meat, if carried on for several weeks, will usually cure the complaint.

4. Lesions of perineum, uterus and ovaries are usually overestimated as causes of back pain and are not common causes of "tired back."

5. Where no cause for chronic pain in the back can be assumed, it should be remembered that a painful back may be used by hysterical patients and malingerers as a protection in their difficulties.

DIET AND "INSULIN" IN THE TREATMENT OF DIABETES MELLITUS.[1]

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THE title of this paper, "Diet and 'Insulin' in the Treatment of *Diabetes Mellitus*" might indicate that an attempt would be made to compare the relative effects of these substances in diabetic treatment. This is not so, however, as they are not properly comparable. Diet is all important in successfully treating the diabetic of whatever severity. "Insulin" is a most useful accessory means of increasing the tolerance of patients suffering from the more severe degrees of the disease. Being the substance normally produced by the pancreas for the metabolism of carbohydrate, it is not indicated in those cases where sufficient insulin is already being produced by the pancreas of the patient except in certain complications of a more or less emergency nature, such as infections.

The possibilities of cure in *diabetes mellitus* seem rather remote. Therapeutic endeavours should then be directed to the restoration of the patient to a maximum degree of health and strength and to enabling him again to earn his living. The under nutrition treatments which have aimed primarily at keeping the individual alive regardless of the cost to his weight and strength, can hardly be held to accomplish a maximal therapeutic effect. The lessons which these methods of treatment have taught us, however, are not unimportant and in some instances patients have been carried on until a new hope arose in the form of "Insulin" treatment. From their results we have learned the dangers of overfeeding in diabetes, the value of rest to the pancreas, the necessity for keeping the urine sugar-free and the importance of a normal blood sugar level in the successful treatment of *diabetes mellitus*.

Others, however, believe that the same objects may be accomplished while using a more liberal diet which permits the patient again to take up his daily round of duties. In severe diabetics this is difficult to attain, but fortunately for these there is "Insulin." Adequate nutrition can only be maintained by the feeding of a sufficient number of calories. Work can only be performed if there is

[1] Read at the annual meeting of the American Therapeutic Society at Toronto in June, 1924.

sufficient energy available. Obviously, then, dietetic treatment with these additional objects in view must resort to quantitative differences in the foodstuffs of a diabetic's menu.

The carbohydrate of the patient's diet must be maintained at a level which does not overtax his damaged pancreas. Protein, which has been shown to be 58% convertible into carbohydrate, also imposes a strain upon the insulin producing mechanism and should not be unnecessarily large in amount. It is known, however, that normally there is a certain minimal breakdown of body protein and this amount must be replaced. In children there is an additional requirement of protein for growth and the breakdown of body protein is more rapid. The former only indirectly increases the work of the pancreas; the latter causes a larger immediate requirement for "Insulin." Since conservative portions of these two important foodstuffs must be fed to the diabetic, it follows that, if he is to be adequately nourished and able to work, a large proportion of his energy requirement must be met from fat. But from fat and certain amino acids of the protein molecule arise the acetone bodies which are largely responsible for the principal danger to the diabetic, acidosis and its result diabetic coma. Of unlimited fat, then, we are very justly afraid, but how much is safe? To Woodyatt, Shaffer, Wilder and others we are indebted for the answer to this problem and it may be said that under suitable conditions ketosis will not develope if one molecule of glucose is being burned while one or two molecules of fatty acid are being oxidized. Several suggested methods of dietetic treatment have grown out of this knowledge. I shall only describe the method which we have found most generally applicable to the needs of our cases, whether treated solely along dietetic lines or in necessary cases with the administration of "Insulin" in addition to diet.

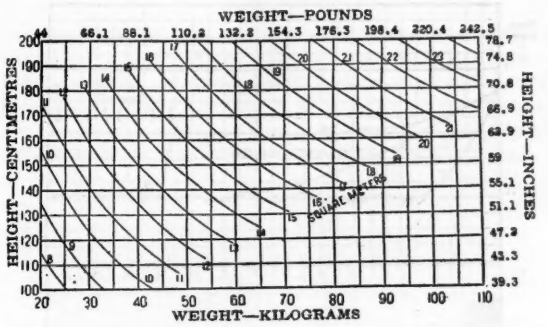
As part of the general clinical examination of a diabetic patient he is weighed and measured. The basal caloric requirement (*M*) of a patient at rest can easily be determined from this data by the use of Du Bois's chart (see Chart I.). Additional energy varying with his occupation will ultimately be required, but for reducing or abolishing the glycosuria the minimal necessary calories should be used. Two-thirds of a gramme of protein for each kilogram of body weight (or 0.3 gramme to the pound) has been shown to be sufficient to satisfy the protein requirement in adults. From the formulæ given below the fat and carbohydrate of the diet can be calculated. The caloric requirement will be satisfied and a safe ratio of fatty acid to the total glucose in the diet is provided.

$$F = \frac{M}{10} - \frac{P}{2} \qquad C = \frac{M - 10P}{30}$$

The actual formation of a menu from the dietetic prescription thus produced is a subject discussed elsewhere. On such a diet the patient is observed for a period of a week or ten days or less, according to the severity of his disease and daily quanti-

CHART I.

SURFACE AREA CHART, SQUARE METRE (DU BOIS).



CALORIC REQUIREMENT PER SQUARE METRE OF BODY SURFACE (AUB-DU BOIS).

Age—Years.	Males		Females.	
	Calories.		Calories.	
	Per Hour	Per Day	Per Hour	Per Day
10-12	51.5	1,236	50	1,200
12-14	50	1,200	46.5	1,116
14-16	46	1,104	43	1,032
16-18	43	1,032	40	960
18-20	41	984	38	912
20-30	39.5	948	37	888
30-40	39.5	948	36.5	876
40-50	38.5	924	36	864
50-60	37.5	900	35	840
60-70	36.5	876	34	816

Calculate requirement in children according to weight. Up to
10 kilograms—55 calories per kilogram.
10-15 kilograms—50 calories per kilogram.
15-20 kilograms—50 calories per kilogram.

Formulæ for calculating food requirements in grammes from Caloric Requirement (*M*).

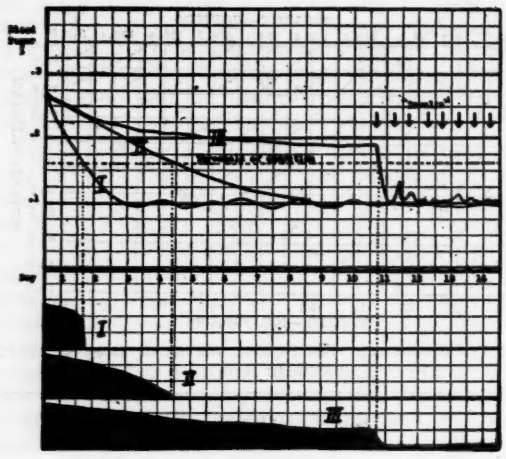
P = 0.66 grammes per kilogram body weight.

$$C = \frac{M - 10P}{30} \qquad F = \frac{M}{10} - \frac{P}{2}$$

Example: Male, aged fifty-five years; height, 71 inches; weight, 154 pounds; surface area, 1.88 square metres (1.88 × 900 = 1,692 calories). Protein = 47, fat = 146, carbohydrate = 40.6 grammes.

tative estimations of the urinary glucose output are made. Estimations of the fasting blood sugar levels are also highly desirable, though perhaps not indispensable at this stage. The patient with a high tolerance for glucose promptly becomes sugar free and his blood sugar level falls sharply (see Chart II.). With a lesser tolerance the glycosuria persists for a longer period and the blood sugar is abnormally high for a still longer period. These considerations will govern to some extent the time at which an increase in diet may be prescribed. From the standpoint of a recovery of tolerance for carbohydrate consequent upon the relief of the pancreas from overwork, it is desirable to avoid too rapid increases in the diet. The time elapsing between the patient becoming aglycosuric and of increasing the diet may vary from two days to two weeks or even longer in the individual case. As protein has no especial value in furnishing the energy required for

CHART II.



Hyperglycæmia and glycosuria in I. mild, II. moderately severe, III. severe diabetics under treatment. (Diagrammatic.)

work and indeed on account of its high specific dynamic action is inferior to carbohydrate and fat in this respect, our first increases in diet are made up from the latter substances, five grammes of carbohydrate and twenty grammes of fat being a usual increase. After an interval if the patient is aglycosuric and free from hyperglycæmia, this increase in diet may be repeated and a third similar increase may follow the second, if conditions appear satisfactory. In the more severe cases careful consideration should be given the question whether more food is actually required for work, as it is always inadvisable to overfeed such patients. Should it be necessary to increase the diet further, it will be found desirable to make the next rise eight grammes of protein and ten grammes of fat. As protein has less antiketogenic power than carbohydrate, in order to avoid an excessive proportion of fat and carbohydrate relative to the protein in the diet, ten instead of twenty grammes of fat are used.

Not less important than the actual treatment of the patient is the training he should receive. It is comparatively easy to find the suitable diet, but prolonged successful treatment of a patient is dependent to a very large extent upon his intelligent cooperation. Few diabetics can learn too much about their disease. Careful education in the fundamental facts about his disease and the reasons for various steps in his treatment, free discussion with him of the results of certain steps in his treatment, free discussion with him of the results of certain procedures as applied to his case and careful training in the dietary management of his disease are essential. Special hygienic precautions should be emphasized. In addition, all patients should learn to test the urine for sugar and for diacetic acid and employ the tests regularly. Records of the results should be preserved and presented to the physician at regular intervals.

The use of "Insulin" introduces additional difficulties in the treatment of the diabetic. Additional care must be taken of the diet. Treatment of diabetics with "Insulin" on anything but weighed diets is an absurdity which needs only to be mentioned to be condemned. The care and use of the hypodermic syringe, the suitable dose, the symptoms, dangers and treatment of hypoglycæmia are additional information which must be taught the patient; while the maintenance of a normal blood sugar level in the patient under "Insulin" treatment is distinctly more difficult than in the patient who can be treated by diet alone. One can never hope to imitate by mechanical means the delicate method of control by which in normal circumstances the insulin production is balanced against the carbohydrate assimilated. In this sense "Insulin" does not replace the internal secretion of a damaged pancreas; it only supplements the activity of the remaining islet cells. It is fallacious to suppose that harmful dietetic measures may be tolerated or their effects overcome by the subsequent administration of a larger quantity of "Insulin." In reality the complete diabetic is exceedingly difficult to treat and the results are most unsatisfactory. The higher the remaining natural tolerance for carbohydrate, the easier "Insulin" treatment becomes and in the severe cases of human diabetes conservation of the little remaining tolerance is imperative.

The objects of treatment of the severe diabetic are the same as in the milder type of the disease, namely, provision of sufficient food for the patient to live and to work and at the same time to maintain the blood sugar at a normal level. Those patients who cannot efficiently metabolize a sufficient amount of food, when placed on a basal diet will continue to show glycosuria and hyperglycæmia (see Chart II.). These patients require additional "Insulin." The amount of sugar excreted after the first few days will become practically constant and on the average output we calculate the dose of "Insulin" required. The glucose excreted in grammes divided by 1.5 gives the number of units of "Insulin" required in each day to make the patient aglycosuric. Subsequent increases in the diet may be made as in the case of a patient treated by diet alone. When a patient becomes sugar-free under "Insulin" treatment, the cessation of overstrain of the pancreas may or may not result in an increase in ability to metabolize carbohydrate. This should not be construed as a true increase in tolerance for carbohydrate due to "Insulin"" treatment. The same condition occurred under dietetic treatment before we had "Insulin." It is simply a release of the latent or submerged tolerance under the influence of rest, is limited in extent and occurs during the first three weeks to three months of treatment. True increase in tolerance which is sometimes seen in the young, is associated with actual new growth of pancreatic tissue and is progressive over longer periods of time. Should latent tolerance be present, increases in diet may be made without additional "Insulin" and

the initial dose may even be decreased. In many severe and long-standing cases, however, this does not occur and three or four additional units of "Insulin" are required to metabolize the usual increase in food.

"Insulin" possesses two actions more or less distinct; one of which causes an increase in the amount of carbohydrate metabolized; the other a lowering of the blood sugar level. The first exerts a wholly beneficial influence, but the latter, if excessive, is accompanied by the utmost danger. When "Insulin" is injected, there follows a fall in the sugar content of the blood. If the "Insulin" be given at appropriate intervals, it may be made to neutralize the excessive postprandial rise in blood sugar level which occurs in diabetes and may thus tend to make the blood sugar remain constant. For this purpose the "Insulin" required is divided usually into two equal doses and administered within the half hour preceding breakfast and evening dinner. In a few cases where the necessary dosage is ten units or less, it may be satisfactory to give the "Insulin" in one dose daily. In severe cases requiring upwards of forty-five units a day, three or sometimes more doses are required to maintain a normal blood sugar level and freedom from glycosuria.

With unbalanced administration of "Insulin" and food assimilation, whether because of increasing tolerance, vomiting, failure of food absorption, diarrhœa or with excessive exercise, excessively low blood sugar levels may be encountered. These are accompanied by a series of symptoms and signs which we have called hypoglycæmia (often miscalled "Insulin" shock). In a general way the severity of the condition, the clinical signs and the relative depression of the blood sugar level parallel each other and one may judge of the treatment required from the clinical evidence.

TABLE.—SYMPTOMS AND SIGNS ASSOCIATED WITH VARYING BLOOD SUGAR LEVELS.

Blood Sugar Levels (Percentages).	Symptoms and Signs.
0.08 to 0.07	Uneasiness, nervousness, tremulousness, weakness, hunger, rise in pulse rate
0.07 to 0.055.. ..	Anxiety, excitement, faintness, emotional disturbances, incoordination, sweating, vertigo, diplopia
0.055 to 0.04.. ..	Marked excitement, emotional instability, sensory and motor aphasia, dysarthria, deafness, delirium, disorientation, confusion, delusions, bradycardia
0.035	Unconsciousness, lost or increased reflexes, increased muscle tonus, tremor, irritability

Patients with mild premonitory symptoms need no treatment if the next meal is near at hand or, if necessary, the condition may be controlled by the juice of an orange or a small lump of molasses chewing candy. With the more severe symptoms a tablespoonful of corn syrup, fifteen grammes of glucose or other rapidly assimilated carbohydrate should be administered and repeated if relief is not experienced within ten minutes. Should a patient be unable to swallow, glucose must be given intravenously in 5% to 50% solution; twenty-five to fifty grammes of glucose are required.

Though one cannot agree that daily fasting and postprandial blood sugar estimations are either necessary or even desirable in the treatment of a patient, since the effect of altering a diet by small amounts is not certain for several days, there is no doubt that for continued successful treatment occasional fasting blood sugar estimations are necessary to detect any tendency to hypoglycæmia or hyperglycæmia. No patient should be discharged from immediate observation until it has been demonstrated that he can maintain a normal fasting blood sugar level on an adequate diet with or without "Insulin." In the earlier stages of treatment blood sugar estimations are useful in determining the size of an increase in diet which may be allowed. With a fasting blood sugar level of 0.08% an increase of ten grammes carbohydrate and forty grammes fat will cause a rise of the fasting blood sugar not higher than 12%. Among patients who become sugar free on their basal diet only after several days, there will be some whose fasting blood sugar will not fall below 0.14%. These will require "Insulin" for all additions to their diets temporarily, if rest to the pancreas increases their tolerance or permanently in cases of long duration. "Insulin" will also be required to lower the fasting blood sugar of such patients to normal in the proportion of two units for each 0.01% above the normal fasting level.

With the increasing duration of life and the excessive consumption of high carbohydrate foods, it is certain that diabetics will increase in numbers. With improved medical facilities, increased appreciation of the significance of symptoms by the public and with the increasing popularity of life insurance and periodical health examination schemes we may expect patients to consult their physicians at earlier stages of the disease. For these the prospect of conserving a tolerance for glucose is better the earlier they are placed under suitable treatment and it seems possible that the relative percentage of those requiring "Insulin" will fall in proportion as all diabetics are adequately treated.

Nevertheless acute and severe diabetes will occur; acute infections, lack of care, failure to obey instructions and other conditions will damage tolerance from time to time and "Insulin" will always be needed, however much early dietetic treatment may reduce its applicability. Much "Insulin" is now being wasted on patients in no way requiring it by practitioners whose ideas on diet are exceed-

ingly or even excessively liberal and to whom a little sugar in the urine is of no consequence. Though at an earlier period it was necessary to limit the number to whom "Insulin" was distributed on account of difficulties of manufacture, this is not now the case. "Insulin" can now be produced in excess of any possible requirement. The problem at present and of the future is to have it used in the best interests of the patient.

Summary.

The treatment of *diabetes mellitus* with diet and "Insulin" requires the provision of sufficient protein, fat and carbohydrate in order to live and to work. A method of calculating this requirement for the individual patient is presented. Should the patient be unable to remain free from hyperglycæmia and glycosuria on this diet, "Insulin" is necessary to supplement the insulin production of the pancreas. It is emphasized that "Insulin" administration is to be regarded as supplementary treatment and this is not to be confused with artificial replacement of the natural carbohydrate tolerance. The symptom complex hypoglycæmia is discussed and its treatment noted.

THE TREATMENT OF ACIDOSIS AND COMA IN DIABETES MELLITUS.[1]

By DUNCAN GRAHAM, M.B. (Toronto),
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ACIDOSIS and coma have been and in cases improperly treated still are the dreaded complications of *diabetes mellitus*. Thanks to the recent contributions to our knowledge of the nature of the disease *diabetes mellitus* and of proper methods for its dietetic treatment and the introduction of "Insulin" in the therapy of the disease, deaths from diabetic acidosis and coma should be the exception and not the rule. However, as long as patients suffering from *diabetes mellitus* are improperly treated or refuse to follow the proper treatment outlined for them or severe infections complicate the disease, cases of diabetic acidosis and coma will continue to occur and death result if appropriate treatment is not given.

In *diabetes mellitus* the body is unable to metabolize the necessary amount of carbohydrate due to an inadequate supply of insulin. As a result, fats are incompletely oxidized and acids, mainly aceto-acetic and β-oxybutyric, accumulate in the blood, producing a state of acidosis or even coma. Acidosis also occurs in non-diabetics suffering from starvation from any cause, following strenuous exercise, after severe attacks of vomiting and diarrhœa and in severe infections and is relieved by the administration of an adequate amount of fluid and glucose. "Insulin" is not necessary. It is, therefore, important to determine, if possible, the underlying cause of the acidosis before beginning treatment.

[1] Read at the annual meeting of the American Therapeutic Society at Toronto, in June, 1924.

The prevention of diabetic acidosis by the giving of an appropriate diet, with "Insulin" when necessary, should be our aim in the treatment of the diabetic patient. As the clinical signs for the early diagnosis of a mild degree of acidosis are too indefinite, the examination of the urine for acetone bodies should be our guide. When a positive reaction for aceto-acetic acid, as indicated by the ferric chloride test, appears in the urine of diabetic patients, precautions should be taken.

In untreated cases of *diabetes mellitus* in which the urine shows a positive ferric chloride reaction without clinical signs of acidosis, the glycosuria and ketosis usually clear up under rest in bed on a basal diet containing a proper ratio of protein, fat and carbohydrate and a liberal quantity of fluids, as outlined in the preceding paper. In more severe cases in which the glycosuria persists and the ketosis is not controlled by rest, dietetic treatment and fluids, the administration of a sufficient quantity of "Insulin" to control the glycosuria is necessary.

In diabetic coma or in the stage of impending coma "Insulin" has proved to be a specific in treatment, but it must be used in conjunction with former methods of treatment. The patient should be at rest in bed with plenty of hot water bottles or other means to keep the body warm. As dehydration is present in all cases of acidosis at this stage, fluids must be given in large quantity. A patient should receive at least four litres of fluid in the first twenty-four hours. If the patient is conscious, fluid in the form of hot tea or coffee, broths and fruit juices may be given by mouth, one litre in the first hour and then half litre per hour; if he has been vomiting or is in a comatose condition, a stout duodenal tube should be inserted and fluid given through the tube. As constipation is the rule in coma and large accumulations of fæces are often present in the intestine, an enema should be given followed by thirty to sixty cubic centimetres of castor oil by mouth or duodenal tube and eight hours later by a saline purge of sodium or magnesium sulphate. In coma cases where vomiting is a prominent early symptom, causing marked dehydration, it may be advisable to forego the use of castor oil and begin the administration of fluids by the Murphy drip method as soon as the bowels have been evacuated by an enema. In the earlier stages of impending coma the patient should receive from twenty to forty units of "Insulin" subcutaneously. If a case is first seen in a private house a dose of "Insulin" should be given immediately and the patient sent to a hospital if at all possible. The urine should be examined for sugar and acetone bodies and a retention catheter inserted, if necessary, in order that the urine may be examined every hour. A sample of blood should be taken for the estimation of the blood sugar, the determination of the carbon dioxide combining power and possibly the percentage of acetone bodies. Information gained from the chemical examination of the blood is a most reliable guide for the future treatment of the patient. If laboratory facilities

are not available, one must depend upon the hourly examination of the urine for the presence of sugar and on the clinical signs and symptoms of the patient to guide him as to when and how much "Insulin" is to be given. Should the condition of the patient not improve by the end of the first hour, a second dose of twenty units of "Insulin" should be given subcutaneously. If the patient is in coma when first seen or is not improving under the subcutaneous injection of "Insulin," an intravenus injection of from forty to one hundred units of "Insulin," combined with fifty cubic centimetres of a 10% solution of glucose is indicated, as a rapid effect from the "Insulin" is particularly necessary at this stage. In order to prevent the development of hypoglycæmia following this, the intravenous injection of the glucose solution should be continued until a litre has been given. The injection should be given slowly, at the rate of ten cubic centimetres a minute, but if the pulse increases in rate ten beats per minute, it should be temporarily discontinued and cardiac stimulants given if necessary. Should little or no signs of improvement in the condition of the patient follow the first intravenous injection of "Insulin" and glucose, it should be repeated at the end of two hours: if distinct improvement is present at the end of two hours, the second dose of "Insulin" may be given subcutaneously and glucose solution by the mouth—one gramme for each unit of "Insulin." With the disappearance of signs and symptoms of coma and severe acidosis, the dose of "Insulin" is decreased and the interval of dosage prolonged, the patient continuing to take the required amount of fluid and also glucose by the mouth. Approximately forty units of "Insulin" *per diem,* given subcutaneously in divided doses, is necessary to control a patient recovering from coma. This treatment is continued, the glucose being gradually reduced and the patient encouraged to take regular nourishment by mouth until he is receiving a basal diet, with or without "Insulin" to control the glycosuria.

Authorities are agreed as to the markedly beneficial effects of "Insulin" in the treatment of coma and that early treatment with "Insulin" is extremely important. There is not general agreement, however, as to the best method of "Insulin" treatment. The use of "Insulin" in the treatment of severe acidosis and coma may be compared with the use of antitoxin in the treatment of diphtheria. In both conditions a small dose given early is more effectual than a large dose given a few hours later. As it is not possible to select cases for early treatment and as the exact amount necessary for the treatment of individual cases is impossible to determine, it is better that the first dose be too large rather than too small, for later treatment may be ineffective. As the blood sugar is usually high in diabetic coma, some authorities believe that with this protection against the production of hypoglycæmia sufficient "Insulin" to combat the acidosis and coma can be given with safety. This may be possible if one has had a wide experience in the treatment of cases of diabetic coma. Unless one is very familiar with the clinical signs and symptoms of diabetic coma and laboratory facilities are available for the chemical examination of the blood, the administration of "Insulin" in sufficient quantity to be effectual in the treatment of severe acidosis and coma is a dangerous procedure, unless glucose is given simultaneously to prevent the development of hypoglycæmia. The presence of sugar in the urine on hourly examination is not an infallible guide as to the safety of another dose of "Insulin" when it is given at intervals of an hour or two as it is necessary in the treatment of coma. The action of the previous dose is not exhausted in this short period and when combined with a second dose may produce a hypoglycæmic reaction despite the fact that sugar was present in the urine at the time of giving the later dose. This reaction, if severe, may be interpreted as a relapse of the condition of coma and have a fatal termination unless glucose is given immediately.

On account of the recognized deleterious effect of an excess of carbohydrate on the pancreas of the diabetic patient, objection has been raised by some against the use of large quantities of glucose in the treatment of coma. When it is remembered that the majority of cases of coma have had a high blood sugar for some time before its onset and that the present object of treatment is the immediate relief of the acidosis, the untoward effects of an excess of glucose on the pancreas for a few hours or even days is not of great significance. Further, the administration of large quantities of glucose is of value in the treatment of coma not only by preventing the dangerous effects from the large doses of "Insulin" necessary in treatment, but by diminishing the acidosis. The diuretic effect of glucose increases the output of water and the excretion of acetone bodies by the kidney. Its combustion by "Insulin" supplies energy and leads to the more complete burning of fat and protein, thereby limiting the production of ketones in the blood.

For many years alkalis have been recommended in the treatment of diabetic acidosis and coma. Recently there has been considerable divergence of opinion as to their usefulness, some contending that they were of little or no value, while others, believing in their value, employed them in treatment. In the past it is certain that too large doses of alkali were given and in many cases by the production of a state of alkalosis did more harm than good. In diabetic acidosis the alkali reserve of the blood, determined by its carbon dioxide combining power, falls below the lower level of normal, 53 volumes % to below 15 volumes % depending on the severity of the acidosis. Adequate "Insulin" and glucose administration, by the burning of the ketones present in the blood and tissues, allows the alkali reserve to return to the lower limit of normal in the majority of cases. In some cases, however, despite the removal of ketones from the blood, the alkali reserve remains at a low level. It is felt that in these cases the administration of alkalis is necessary. As alkalis should be given early, every

case of diabetic coma should receive a moderate amount of alkali at the beginning of treatment. A dose that may be given with safety is twenty grammes of sodium bicarbonate for each thirty-eight kilograms of body weight. If the carbon dioxide combining power of the blood does not return to normal, further alkali treatment is indicated.

Brief reference only will be made to the importance of infections as a complication of diabetic acidosis and coma as the question of infections as a complication of *diabetes mellitus* will be discussed in the next paper. The development of an infection and of acidosis and coma are two of the danger points in the life of the diabetic. Even mild infections lower the carbohydrate tolerance of the diabetic and predispose him to the development of acidosis and coma and their presence in cases of acidosis and coma makes the prognosis much more serious. It is, therefore, important in all cases of *diabetes mellitus* undergoing treatment that a careful search be made for possible foci of infection and that these be eradicated after the diabetic condition has been controlled by appropriate dietetic and "Insulin" treatment. In cases of acidosis and coma complicated by infection, treatment must be directed first to the control of the acidosis and later to the control of the infection for experience has shown that in diabetes an infection only responds favourably to treatment when a normal metabolism has been established by appropriate dietetic treatment.

THE TREATMENT OF OTHER COMPLICATIONS IN DIABETES MELLITUS.[1]

By A. Almon Fletcher, M.B. (Toronto),
Toronto.

The diabetic patient is remarkably liable to the development of various complications. Heretofore these complications have played a major part not only in the tendency for diabetes to grow more severe, but also in its high mortality. This liability is particularly marked in the untreated or unrecognized case or when treatment has been imperfectly carried out and the greatest insurance against the occurrence of complications is the establishment of an adequate carbohydrate metabolism and normality of the sugar in both urine and blood. With the advent of "Insulin" and the careful dietetic control made necessary by its use, this liability is much diminished. But, with the fall in the mortality of this disease even in its most severe form, there is now a constantly increasing diabetic population which presents special medical problems in the treatment not only of its characteristic complications, but also of the various bacterial invasions and surgical emergencies incident to any population.

Infections.

Of the complications infections are the most important and their occurrence in a diabetic patient should always be considered serious. It seems true that the well treated diabetic, even though under-nourished, is not more liable to infections than the healthy person. Patients under regular "Insulin" treatment maintain as a rule that colds, for example, are not more frequent than before the onset of the disease; but once an infection becomes established, carbohydrate metabolism is upset and the infection tends to run a severe and prolonged course if some support to the disordered carbohydrate metabolism is not given. The persistence of boils in a diabetic until the urine is rendered sugar-free is a common experience and shows the intimate association between sugar metabolism and resistance to bacterial infection. The disturbance in carbohydrate metabolism can be explained both on the basis of increased "Insulin" requirement and decreased insulin production on the part of the pancreas. On the one hand metabolism is raised, varying with the severity of the infection and part of this increased heat production should be borne by carbohydrate combustion. On the other hand the infection results in toxic injury to the islets of Langerhans and there is a marked lowering in the amount of insulin produced. Sometimes during the course of an infection a comparatively mild case will present a clinical picture of total diabetes. The decrease of insulin production is early manifest both in raised blood sugar levels and ketone intoxication, often going on to acidosis and coma if "Insulin" is not given. In addition the toxic injury to the islet tissue may result in permanent damage with decreased power of carbohydrate utilization after the infection has subsided.

With the onset of any frank infection "Insulin" should be administered, both to tide the patient over the immediate emergency and to guard against possible damage to the islet tissue. Ordinary colds or upper respiratory infections should, if possible, be prevented or prompt measures instituted for their treatment. The patient should be at rest in bed, "Insulin" administration started or increased and an attempt made to control the rise in blood sugar and the excretion of glucose. Severe or general infections call for most careful measures. If necessary "Insulin" may be administered up to forty units every three hours during the day, this amount being determined by the sugar and ketone excretion. The production of small amounts of ketones is unavoidable, but a well marked ketosis with a strongly positive ferric chloride reaction in the urine demands large doses of "Insulin" and in most cases increased carbohydrate administration. Reduction of the blood sugar level alone under such conditions is not sufficient and carbohydrate combustion must be materially increased to combat the disturbance in fat metabolism. The carbohydrate of the diet should be increased and if the blood or urinary sugars have fallen while the ketone excretion persists, glucose should be given along with more "Insulin." It is remarkable how much "Insulin" such patients may require and tolerate. It would seem as if "Insulin" had a lowered carbo-

[1] Read at the annual meeting of the American Therapeutic Society at Toronto in June, 1924.

hydrate value in the presence of severe infection. In a fever of any duration protein may be increased to one gramme or even one and a half grammes per kilogram. Fats are usually not well borne and should be decreased. In the severe infections the picture is often that of impending coma. Besides hyperglycæmia and severe ketosis we have to consider acidosis and dehydration. The need for bicarbonate of soda may be determined by the carbon dioxide combining power of the plasma or by the onset of the symptoms of acidosis. With the loss of fluid due to the fever and diuresis of hyperglycæmia, sometimes combined with the vomiting of acidosis or toxæmia, dehydration may be extreme and restoration of body fluid is urgent. Two or three litres may be given in the first twelve hours and the administration of fluid may be carried out if necessary by stomach tube, by rectum, interstitially and intravenously.

Striking clinical results are often seen. Pneumonia, influenza and other general infections appear to run their usual course. Sometimes the "Insulin" requirement may be as high as one hundred and fifty or two hundred units daily. In any case of impending or actual coma infection is to be suspected. A patient may be first seen with cold and cyanosed extremities and a markedly subnormal temperature. An infection such as bronchopneumonia is easily overlooked especially in the absence of fever due to failure of carbohydrate and fat metabolism. Under these conditions fever although due to the infection may develope only after "Insulin" is injected and the rapid rise in temperature may even be accompanied by a chill.

Local infections, when possible, are best treated surgically. The diabetic is a poor subject to carry a focus of infection, but these operations are not to be undertaken lightly. The diabetes must be well under control beforehand. In the case of the teeth it is well to remove not more than two at a time. Much harm to the islet tissue may be done by the removal of several infected teeth at one operation.

Tuberculosis, although decreasing in its incidence among diabetics, is still met with. Not infrequently it developes before the discovery of glycosuria. Recognized early the tuberculous process may be entirely arrested. In these patients special consideration is given to the adjustment of diet and any measure which will result in further undernutrition, is to be avoided. While it has not been found necessary to furnish the large diets of 3,500 or 4,000 calories sometimes recommended, the patient at rest in bed may well be allowed 700 or 800 calories above the basal metabolic requirement. This will usually result in material increase in weight. The total calories may be controlled to bring the weight to average for the height and age of the patient, but further increase in weight does not appear necessary. Instead of the usual two-thirds gramme of protein per kilogram of body weight it has been found advisable to increase this to one gramme and a half on account of the increased protein breakdown in the presence of infection and on the general experience that adequate protein is beneficial in combating tuberculous infections. With this one gramme and a half of protein per kilogram a positive nitrogen balance will, as a rule, be observed. "Insulin" should be pushed until the urine is free of sugar. Low blood sugars would be desirable but with high diets and high "Insulin" dosage normal blood sugars cannot always be established without the occasional occurrence of hypoglycæmia. Two cases of tuberculosis and severe diabetes have been treated on these lines and have shown very marked improvement. In other cases the disease was much more advanced, but the progress of infection may be checked.

Gangrene.

The development of complications calling for operative interference is frequent among diabetics. Gangrene is only too common, but besides this many infections require surgical intervention. In the diabetic, for example, the gall bladder is more frequently diseased than in the non-diabetic and the frequent occurrence of foci of infection is well recognized. Operations in the past have always run a high mortality. The wounds heal badly and readily become infected. Besides this the operation and the anæsthetic both appear to make the diabetes worse. An operation, uncomplicated by infection, may convert a mild case into one of coma and it is important to support the carbohydrate metabolism under this increased strain. "Insulin" in small doses, such as ten units three times a day, may well be given for three or four days following the operation as a matter of routine. This amount will often have to be increased and when necessary additional glucose given to guard against hypoglycæmia. It is usually advisable to give "Insulin" and glucose before the operation as a further precaution against ketosis, probably because the carbohydrate stores protect the liver from the toxic damage of the operation and the anæsthetic. In this connexion it should be mentioned that the anæsthetic of election is gas and oxygen. Well prepared beforehand and care taken to support the carbohydrate metabolism afterwards, the diabetic stands an operation well, provided there is still good function of both heart and kidneys. Gall stones, for instance, have been removed in moderately severe cases without any delay in the healing of the wounds and with subsequent benefit to the patient.

In the case of gangrene, as in most complications, prevention is still the most urgent consideration, but the outlook need no longer be considered grave. It is well to remember that arteriosclerosis may be only one of several causative factors, for often enough the surrounding tissues show considerable circulation. The exciting cause of the gangrene is often either neglect of the feet or neglect of the diabetes and often both. For this reason, conservative medical treatment should be carried out in early cases, rest in bed, elevation and protection of the part and along with "Insulin" administration

the establishment of normal blood sugars and a diet sufficient to support a feeble cardio-vascular system. Small areas of gangrene may be checked and end only in superficial sloughing, if the X ray picture shows an absence of sequestrum formation. Operation, if necessary, may be safely carried out even in elderly patients; but in these cases the wound will heal slowly, not because of the diabetes, but because of the arteriosclerosis.

Neuritis and Arthritis.

Diabetes is often complicated by pain which may be of the nature of neuralgia, neuritis, arthralgia, frank arthritis, spondylitis, lumbago, fibrositis and so on. Not infrequently the pain is an early feature of the case. In the majority of these conditions if there is no focus of infection, the pain is relieved by treatment of the diabetes, but in a few cases the patient gets little or no relief. It is to be emphasized that the relief of glycosuria is not a sufficient measure in this treatment. Many of these cases have a high renal threshold and a high blood sugar. It has not been an unusual experience to observe the persistence of pain after the urine has been rendered free of sugar, but relief of pain when normal blood sugar levels have been attained. When this can be done by dietetic means there is no advantage to be gained in the use of "Insulin," but failing this "Insulin" should be given in adequate dosage.

Multiple neuritis is more refractory to treatment. The most severe lesion of this type that I have seen, was in a patient with a renal threshold of over 0.3% and at that the urine not always sugar-free. In this condition the process may perhaps be checked by treatment, but little prospect of improvement in the motor or sensory changes can be offered.

Cardio-Vascular Complications.

The lesions of the cardio-vascular systems are also difficult of treatment. Arteriosclerosis, *angina pectoris* and myocardial weakness are frequently observed, especially in the untreated case, and accidents such as coronary thrombosis and cerebral apoplexy are not uncommon. In severe diabetes ideal conditions of blood sugar control cannot be established and further myocardial degeneration may take place with the gradually increasing signs of cardiac insufficiency. The occupation for such patients after treatment has been carried out presents a difficult problem and, indeed, the advisability of allowing any patient suffering from severe diabetes to return to a laborious occupation or heavy house work may well be questioned. As is often pointed out, the symptoms of myocardial weakness may be brought on or aggravated by low diets, especially by insufficient protein. These patients may be given a gramme to a gramme and a half of protein per kilogram of body weight and they undoubtedly feel better when this is done. On the other hand, weight should be reduced when obesity is present and while this reduction may also aggravate symptoms for a time, there is an increase in the cardio-vascular efficiency when the weight has fallen.

Eye Lesions.

The eye lesions, such as optic neuritis or retinitis, can usually be checked and many such conditions have shown considerable improvement by the establishment of treatment.

The relationship between these complications and the disease itself is by no means clear. Cases with high blood sugar and glycosuria persisting over considerable periods of time are not infrequently met with and many of them have been neither the subject of any severe infection nor evident degenerative lesion. But as a group these are the cases which show complications and it is difficult to avoid the conclusion that excess sugar in the blood is directly associated with their development.

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THE MANAGEMENT OF GASTRIC AND DUODENAL ULCERS.

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The debate which has been carried on consistently for so many years as to whether gastric and duodenal ulcers are the property of the surgeon or the internist, is gradually solving itself as the result of a more thorough appreciation of the physiological principles underlying digestion. It is not so long ago that diagnoses of gastric or duodenal ulcer or of gastric cancer were made as a result of an analysis of the gastric content following the classical Ewald test meal. In recent years this means of diagnosis has been relegated to antiquity, because of the work of Rehfuss and his development of the standardized analysis of gastric contents, known as the fractional test meal. Using this method he has demonstrated alterations in the free and combined acid of the gastric content in healthy students which were not dreamed of previously and has shown that these bear a constant and definite

relationship to the various phases of gastric digestion.

In the early development of the surgical therapy of peptic ulcer operation was never undertaken, except when there was incontrovertible evidence of obstruction. As we all know, a gastro-enterostomy in such a condition gives most brilliant results. As the evolution of abdominal surgery progressed, the clinical diagnosis of ulcer was made by the internist and the case handed over to the surgeon who automatically did a gastro-enterostomy without confirming the presence or absence of an ulcer. After such a procedure the results left much to be desired, as in many cases the lesion was extrinsic to stomach or duodenum; they were not at all comparable with the excellent results of a gastro-enterostomy in the presence of an organic obstruction. From these facts the conclusion was reached that the excellent original results were explained by the presence of the obstruction. Thus pyloric exclusion with all the various technical procedures became the fashion and yet despite this the results left much to be desired. In view of the above we arrived at our present idea of the underlying cause of gastro-intestinal symptoms, namely interference with motion, due to a spasm or organic obstruction in the region of the ulcer. To-day no surgeon would contemplate a gastro-enterostomy or other operation for ulcer without first convincing himself that there was a definite organic ulcer present.

With this evolution in the purely technical side of ulcer therapy has arisen the necessity of interpreting gastro-intestinal symptoms as altered normal physiological phenomena. It is felt that all symptoms of gastro-intestinal disease, no matter where located, are the direct result of interference with normal physiological motion, that is delay in the normal onward flow of gastro-intestinal contents. Such may be due to atony of the musculature or to spastic or to organic obstruction. In order to appreciate the factors responsible for normal motion, we must take cognizance of the dual nerve supply of the gastro-intestinal tract. As far as gastric and duodenal lesions are concerned, the dual supply is the sympathetic fibres controlling the pyloric sphincter and the vagus fibres controlling the rest of the musculature in the stomach and duodenum; hence we can immediately visualize lesions causing reflex disturbances, situated elsewhere than in the stomach or in the duodenum. Such areas are of necessity intimately associated with this duplicate nerve supply. Lesions in such sites cause an upset in the normal motion of the pylorus, due to lack of coordination of the vagus and the sympathetic neuro-muscular apparatus in the stomach and the pylorus respectively.

The two regions where such lesions are most often situated are first the biliary tract where we have the sphincter of Oddi controlled by the sympathetic nervous system, as well as the vagus supply to the other portions of the tract; and in the ileo-caecal region where we have the ileo-caecal sphincter, controlled by the sympathetic nervous system and the rest of the gut controlled by the vagus. Clinically we know that lesions in these two areas may produce reflex spasm in the region of the pylorus, thus simulating a true intrinsic gastric or duodenal lesion.

If our hypothesis regarding the cause of symptoms be correct, then motion in the stomach or duodenum can be altered only by an obstruction. Such obstruction must be either due to organic scarring or due to muscle spasm. The spasm may be situated in three areas: in the stomach proper, in the pylorus or in the duodenum. Thus our only clinical method of determining the site of the spasm lies in the interpretation of the symptoms in relation to the intake of food and in relation to the normal rate of flow in the tract. As an example, the most easily demonstrable cause of pylorospasm is the terminal ileal kink. In this lesion the symptoms have a very constant relationship to the ingestion of food which can be interpreted farther afield in relation to the time of day. The clinical history of such individuals suffering from so-called indigestion is that they feel well in the morning and enjoy their breakfast, are not so enthusiastic about lunch and in the afternoon feel wretched and have no appetite for dinner. This we can explain. Because of obstructive phenomenon they are carrying in their terminal ileum breakfast and lunch for an unduly long time, producing a reflex interference with motion in the stomach, causing a lack of normal tonic contraction in which state the stomach exists in hunger. Such an indigestion coming on in this way is most unlikely to be due to a lesion situated intrinsically in the stomach or duodenum. The proof that this lesion is the result of such a mechanical condition is easily demonstrated by asking the individual to refrain from anything but fluids for the noonday meal, following which his indigestion will immediately disappear.

In the duodenal and gastric lesions on the other hand the symptoms are more closely related to the individual meal. That the symptoms are the result of spasm and not the result of the irritation of food passing over an ulcerated surface, as used to be thought, can be proven by the fact that radiographically one can demonstrate an ulcer on the lesser curvature which, having penetrated all the muscular coats, no longer produces muscle irritability and hence no muscle spasm. The individual with an ulcer which has penetrated through the whole thickness of the stomach, will have no symptoms or will have continuous symptoms irrespective of the intake of food. The latter are due to a perigastric inflammatory reaction, the result of the penetration of the ulcer into adjacent structures. One of the fairly constant clinical findings in such an ulcer is the immediate relief which such patients experience on lying down, and the sudden reappearance of their pain on assuming the erect posture. This is possibly the result of a drag by the stomach on involved gastro-hepatic omentum.

Furthermore, there is a very definite difference in the symptoms of individuals suffering from a gastric ulcer as compared with those suffering from a duodenal ulcer. The duodenal ulcer individual

approaches the meal with the so-called "hunger pain." This hunger pain has been demonstrated by Carlson to be due to a hypertonicity of the whole gastric musculature. The relief from the pain in duodenal ulcer which follows the ingestion of food, is due to a relaxation of this spasm of the whole stomach. The pain, however, returns in a variable time, to persist until food again enters the stomach; hence the great virtue of the so-called Sippy diet which always leaves some food in the stomach and thus prevents this hypertonic contraction with the resultant pain, lies in the frequent feedings.

The patient with a gastric ulcer, on the other hand, has frequently no distress prior to the taking of food. Because there is not immediately initiated hypertonic gastric contraction, there is no distress immediately on taking food. In a very short time, however, this occurs and it is believed to be due to a spasm set up in the gastric musculature, due to the irritation of the inflammatory reaction about an otherwise normally contracting stomach. This pain will persist until the stomach has emptied itself and assumed a state of rest, *id est* the pain does not persist until food is again taken, as in duodenal ulcer; hence the efficacy of the duodenal tube in the treatment of gastric lesions, as it can allow the stomach to maintain that state of absolute rest which is so essential to the avoidance of further spasm with resultant freedom from pain.

Assuming the above to be true, one can fairly accurately arrive at a diagnosis of duodenal ulcer and a reasonably accurate diagnosis of a gastric ulcer. One must, however, confess that the bizarre character of the symptoms in so many gastric ulcers which I believe are largely the result of perigastric involvement, so confuse the relationship between the intake of food and the occurrence of symptoms that the diagnosis is often uncertain.

Before one can outline dogmatically definite treatment for any disease, it becomes necessary to determine the cause. The ætiology of peptic ulcer is unknown. There is a definite relationship between foci of infection and the presence of ulcer. This, I believe, no one can doubt. Because of the close association of the sympathetic neuro-muscular apparatus with the physiological process in gastric digestion, I feel that environment, fatigue, worries and other emotions must play a part in the persistence of symptoms. The treatment of individuals suffering from ulcer must of necessity include the correction of these two important factors in addition to the therapeutic procedures dealing directly with the ulcer.

In order to determine the cases which must have surgical therapy in addition to dietetic control, we are guided by the following indications: First the stenosing ulcer, causing organic obstruction; second the individual who will not respond to dietetic control; third the individual who responds to dietetic control and remains symptom-free so long as he is on a very severely restricted diet and under ideal environment, whose symptoms immediately return on resuming his normal work; fourth the individual who economically cannot afford the time necessary for proper dietetic treatment, with the possibility of a failure resulting in a further period away from work. The minimum time for medical and dietetic treatment of ulcer is one month in bed, followed by a very markedly restricted work period and a carefully selected and controlled diet for a further two months. This requirement in the case of the ordinary hospital patient with a family to support is very often an economic impossibility. The fifth indication includes the perforated ulcers.

Of these five groups the first and the last leave no room for argument. The other three will present many less patients for surgical therapy if the physician will realize that ambulatory dietetic treatment for ulcer is almost inevitably unsatisfactory.

It will be noted that hæmorrhage has not been mentioned as of itself being an indication for surgical therapy. The cases in which an emergency operation is advisable solely because of hæmorrhage, are extremely rare and in many cases in which an emergency operation for hæmorrhage will be undertaken, no gross pathological lesion will be found.

The technical procedures in dealing with ulcers of the duodenum and stomach vary on fundamental principles, because of the fact that carcinoma of the duodenum is exceedingly rare. Whenever possible the involved tissue in the duodenum should be removed either by the cautery or a knife excision. It is advisable to do a reconstructive type of operation whenever possible after the excision of the involved tissue rather than a gastro-enterostomy which may result in a marginal ulceration.

In gastric ulcer, on the other hand, we have the *bête noire* of carcinoma being engrafted on ulcer as a real possibility. A recent survey of the gastric ulcers in the Toronto General Hospital has been made by Dr. Wookey and he found 18% of the ulcers clinically diagnosed as benign were proven histologically to be carcinomatous. This emphasizes the importance of excising whenever possible the ulcerated area either by means of the cautery or the knife. We feel that the latter when technically possible is more likely to result in thorough eradication of the disease. Such excisions so commonly produce a deformity of the stomach that such should be anticipated by doing a gastro-enterostomy and placing the stoma in such a position as to drain both segments of the stomach, should an hour-glass develope. In certain cases it is safer and technically easier to do a partial gastric resection than to attempt a local resection.

In the large ulcers situated near the cardia on the lesser curvature and in the penetrating adherent posterior wall ulcers in which practically no type of radical excision is technically possible, it is advisable to open the anterior wall of the stomach parallel to its long axis in a segment of stomach opposite the ulcer, cauterizing it as thoroughly as possible and oversewing the mucous membrane. This also serves the additional purpose of putting

that segment of stomach absolutely at rest and by suturing the excision transversely to the long axis, avoids an hour-glass which might follow the healing of the ulcer. Such a procedure should be coupled with a gastro-enterostomy.

Regarding the position of the stoma it is considered that this should be placed near the greater curvature opposite the *incisura* rather than near the pylorus. In instances where the stoma has been placed near the pylorus, we have found a very rapid emptying of the stomach with persistence of distress after meals.

Further, one must not regard any surgical therapy for ulcer as the final treatment of the case. We have been accustomed to place our patients on a very strict dietetic *régime* for a period of six months, each patient on discharge from the hospital being given the following instructions:

After your discharge from hospital the most important factor in securing a good ultimate result is a rigid adherence to the well-accepted, commonsense principles of life, avoiding excesses of any sort, particularly undue fatigue, breaking up the work period by short rests whenever possible, for the first six months never standing when you can sit down and never sitting down when you can lie down. You must, however, not consider yourself an invalid, but by carrying out a fairly strict *régime* for six months you should have an excellent result. You will probably enjoy better health immediately than you have for years, but this is no indication that you can take liberties with regard to irregularities in diet or excessive work, either mental or physical.

Your diet list for these six months broadly should include the following: Soups, any light soup; meats, any of the easily digested meats, sweetbreads, red beef, mutton, lamb, poultry. These for the first two or three months at least should be put through a meat chopper and should be broiled or boiled. Fish, mainly the white variety, fresh water fish preferable; oysters, boiled or raw; eggs, in any form except fried; vegetables, for the first two or three months these should be mashed and strained or put through a food chopper, the easily digestible kinds such as asparagus, spinach, peas, beans, potatoes and carrots. Any of the cereals can be taken, but oatmeal and similar porridge should be cooked thoroughly in a double boiler. Fresh bread should be avoided, but bread a day old may be taken. Desserts, any of the light puddings; fruits, mainly stewed fruits; fatty foods, cream, butter and olive oil; drinks, milk, butter-milk, cocoa, carbonated mineral waters and an abundance of plain water.

The following must be avoided: Rich soups, fried foods, pork, veal stews, hashes, corned meat, potted meat, twice-cooked meat, liver, kidney, duck, goose, sausage, crabs, sardines, lobster, preserved fish, smoked fish, salted fish, salmon, cauliflower, celery, radishes, cabbage, cucumbers, sweet potatoes, tomatoes, beets, corn, salads, bananas, melons, berries, pineapple, hot bread or cakes, nuts, candies, pies, pastry, preserves, cheese, strong tea, strong coffee, alcoholic stimulants and any excessive condiments.

The use of tobacco should be dispensed with for three months and then used in moderation, preferably only after the evening meal, but at any rate never before luncheon.

This diet will allow of a certain laxity after three months, but for the first three months it is considered very important that you follow it fairly rigidly. During the first three months it is essential that you should avoid trying to carry on with three main meals. Rather you should supplement these by malted milk, eggnog with toast or some such light nourishment at ten o'clock in the morning, between three and four in the afternoon and before retiring. If there is any return of pain similar to your former distress, report to your physician at once.

THE TRANSPORT AND DEPOSITION OF CALCIUM SALTS IN THE BODY.

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Calcium is one of the valuable and necessary inorganic constituents of the tissues of the body. Its presence in the blood in normal quantity is essential to good health and without a proper proportion of it in teeth and bones their integrity is threatened. It is not my purpose here, however, to review the work on the metabolism of calcium, but to confine myself to an exposition of how this element is transported in the body and the part that it plays in the formation of bone and of calcareous deposits.

Calcium may be ingested in various forms in our different articles of food and drink, but it is probable that these are practically all converted into the soluble chloride by the hydrochloric acid of the gastric juice. In this form it is absorbed and taken into the blood stream and at first thought it might be conceived to circulate as the chloride. A moment's consideration, however, will show that this is unlikely owing to the large amount of carbonates and phosphates in solution here, which will react to precipitate the calcium as the insoluble carbonate and phosphate. Pauli and Samec have shown that in a colloidal solution containing a protein such as the blood serum does, these salts of calcium are sparingly soluble, however, and the possibility remains that they could thus be carried in the blood. But Barillé has demonstrated a further step, in that these two salts of calcium, the carbonate and phosphate, combine at their formation in the blood into a double salt, calcium carbono-phosphate: $P_2O_8Ca_2H_2 : 2CO_2 (CO_3H_2) Ca$, which is quite soluble in the concentration of carbon dioxide found in the blood, but is precipitated and broken up into its separate constituent salts if the carbon dioxide concentration is lessened.

Wells has analysed many bones and areas of calcification and has found that the proportion of calcium carbonate to calcium phosphate in these areas is fifteen parts of carbonate to eighty-five of phosphate by weight or one molecule of carbonate to three of phosphate. Barillé's double salt on precipitating and breaking up into the separate constituent salts yields exactly this proportion, so that calcium is thus seen to be transported in the blood in exactly the proportions in which its salts are found in bone and calcified areas. It is an interesting fact that in minerals containing calcium carbonate and phosphate the combination of the two is found in proportions similar to the above.

The manner in which calcium salts are taken from the blood to impregnate bone and calcified areas is not definitely proven, but appears to be quite different in the two cases and will be discussed separately.

In bone the calcium salts form two-thirds of the mass. They are all contained in the matrix, with

the organic constituent of which they are so intimately blended as to be indistinguishable in many cases. The ossein, the organic material of the bone matrix, is secreted by the bone cells or osteoblasts as an exoplasm and is similar to the fibres of white fibrous tissue (Schäfer). In a growing bone, especially in rapidly growing specimens such as are found in the fœtus, calcium salts can be seen appearing in the organic matrix immediately upon its formation as a cloud of extremely minute granular particles (Watt). This granular appearance soon disappears as the calcium content increases and the matrix looks clear and glassy and homogeneous.

The main problem involved here is the manner in which the calcium is deposited. The very fine granular beginning of the deposition might indicate either precipitation or secretion as its source. Wells believes the salts are precipitated from the calcium carbono-phosphate which diffuses in from the blood, into a matrix poor in carbon dioxide. Stump, Maximow, Weidenreich and others hold the process to be a secretory function of the osteoblast. In a previous paper I have shown that calcium carbonate precipitated in colloids can be identified readily and separated from the phosphate. Such identification and separation is not the case in bone. I have also shown that there is a strong tendency for calcium deposits in colloids to fuse and this may account for the rapidly developing homogeneous appearance of the bone matrix as it becomes fully impregnated.

Not all bone appears homogeneous, however, for some parts even when fully formed show a granular appearance. Weidenreich has offered an explanation of this in his classification of bone into fibre bone (*Faserknochen*), really calcified periosteum and ends of tendons and ligaments, cartilage bone, (*Knorpelknochen*) which is calcified cartilage, best seen in the primary ossification in fœtal bones and lamellar, plate-like bone (*Schalenknochen*) as seen in the Haversian systems of bone.

The first two varieties are granular in appearance and are to be interpreted as calcifications, the third is more homogeneous and Weidenreich considers it as wholly due to the secretory activity of the osteoblasts.

We know that bone is a tissue that is very active, continually changing, some parts being built up, others removed or altered in response to strain. This activity is especially evident in the change due to growth, for as the long bone increases in size, the interior medullary cavity is coincidentally enlarged. Repair after fractures and operations amply demonstrates this as does also the rarefaction of the bones in a limb kept in a splint. It is generally admitted that the osteoblasts, the bone cells, as well as building the original bones, are responsible for its changes and are continually active.

Now it has been shown that the matrix of bone which is being removed, is in all probability absorbed by the osteoblasts (Galley and Robertson). The lime salts are removed as well as the ossein and, if they were deposited by precipitation, it is inconceivable that these salts can be removed without a reversal of the conditions which brought about their deposition. But such a change of condition in the matrix, implying a great increase in the carbon dioxide content, is unlikely and so the absorption of the calcium salts may be logically ascribed to the activity of the osteoblast. Stump shows that it is reasonable to ascribe to the bone cell the power of removing again the substances of the matrix and looks upon the osteoblasts as aggregations of primitive connective tissue cells which are also osteolytic in function.

The processes of ossein formation and of deposition of lime salts in the ossein, while occurring at the same time in newly forming bone, are apparently at least partly independent processes. The fibrous matrix can be seen in a rapidly growing area of ossification just prior to the appearance of the first fine granules of calcium salts. This is best seen right at the advancing edge of ossification in a fœtal long bone. The calcium granules rapidly increase in numbers and fuse, so that fully formed bone is seen just back of the edge. Whether the lime salts are deposited in or between the fibres is not known, both views being widely held. Further proof of the independence in the two processes forming the bone matrix is seen in such a disease as rickets, where the organic matrix is fully formed, but impregnation with calcium salts is defective.

Foods rich in calcium and in phosphates favour good growth of the skeleton in children, also proper repair of fractures and other injuries of bone, but are often insufficient in themselves to ward off trouble or to effect repair, as is frequently the case in rickets. The beneficial effects of sunlight in this disease through the action of its penetrating ultraviolet rays, leads one to suppose that these rays exert a powerful beneficial stimulus on the bone cells, restoring them to normal power and activity.

It is undeniable that changes in the action of the ductless glands also affect very markedly the growth of the skeleton. Engelbach and McMahon have given good examples of this investigated by means of X ray examination of the bones. Deficient, excessive or altered function of the pituitary gland, the thyreoid or the gonads all have marked effects on the growth and ossification of the skeleton. The most plausible theory as to the action of the hormones of the endocrine glands is that they exert their effect upon the osteoblast, its relative activity being the resultant of the amount and character of the hormones acting upon it at the time.

Another indirect evidence of the activity of the osteoblast may be obtained from the fact pointed out by Wells and others that where great demands for calcium are made upon the body, the bones become rarefied by absorption of some of their tissue to meet this demand. Such occasions arise in pregnancy, the growing fœtus robbing the mother of lime salts and also in cases of pancreatic fistula where there is a steady loss of calcium. Indeed, Wells has come to regard the bones in these cases as a great alkali reserve with its material in a state of flux, to be drawn upon if necessary and replaced whenever possible.

The fact that calcium may be withdrawn thus in cases of need has led me to evolve the idea that there may be a sort of calcium balance in the body between the lime content of the bone matrix, the bone cell and the blood. Where normal quantities are present in the blood the osteoblast may withdraw calcium salts from it and deposit them in the bone matrix. When the blood is poor in calcium, the normal balance or equilibrium is upset and the osteoblast withdraws lime salts from the bone matrix in an effort to raise the blood content and restore equilibrium. This would resemble very closely the carbohydrate balance between the stored glycogen of the liver cell and the amount of sugar in the blood. In this case when sugar in the blood is in excess of the normal amount in ordinary individuals, the liver cell stores up the excess as glycogen to be returned to the blood when its sugar content falls below normal.

In areas of calcification the conditions are somewhat different to those found in bone. We may separate these cases into two different kinds: Firstly calcification of normal living tssues such as occurs in the cartilaginous skeleton preliminary to ossification and secondly, areas of necrosis, areas of fibrosis, in fact any damaged area which becomes calcified.

In the first place it appears impossible to state at present whether cellular activity is responsible for calcification in cartilage. It is unlikely, however, because the calcified area in advance of the ossifying region of the fœtal bone, for instance, contains cartilage cells which are usually looked upon as either dead or dying. They are certainly very much altered from the normal cartilage cell. The calcium salts here are deposited in very evident granules, closely packed and lacking the homogeneous appearance of bone. They occur first in the neighbourhood of the invading vessels. The appearance is more like that of a deposition by precipitation from a slowly diffusing solution.

There is, however, a very close chemical union of calcium and matrix and the matrix of calcified cartilage is in some way altered, for it stains differently to that of the uncalcified areas (Weidenreich). I can corroborate this for other areas also.

In necrosed and damaged areas calcification is undoubtedly of the nature of a precipitation. If one is willing to sacrifice a microtome knife, fairly good sections can be cut without decalcifying. I have examined both by light and dark field illumination specimens of fœtal bone, adult cancellous bone, calcified cartilage and calcified arteries, thyreoid gland, pineal gland and chorioid plexuses. Bast's method of examination was also found very useful. In the areas of calcification the calcium was present in many cases in a crystalline form, in others in dense aggregations of granules. The fracturing of these masses by the knife gave illuminating information as to their structure. In pineal gland and chorioid plexuses round, doubly refracting masses of calcium in concentric layers were seen in every way similar to the colloidal spherical crystals of calcium carbonate which I have studied in precipitations of calcium carbonate in colloidal solutions.

Wells has stated that areas of calcification contain lime salts in the same proportions of carbonate and phosphate as bone. I believe from what I have seen that this is not true for some small masses, such as those in the pineal gland and chorioid plexuses where the appearances are distinctive of calcium carbonate deposition. The bodies seen here to my knowledge can only be formed in one way and that is by precipitation. Their presence is thus an evidence of the manner of deposition in calcified areas.

Klotz has also demonstrated that in arteries the first appearance of calcium deposition in areas of degeneration or necrosis was by precipitation of calcium in the form of insoluble salts of fatty acids which had been produced in these areas. These salts are subsequently slowly changed into carbonates and phosphates which fuse in typical crystalline masses.

These masses of lime salts found in the tissues are always in areas either dead or of very low grade activity where cellular activity in their deposition can be ruled out, leaving only precipitation as the method of deposit. I have examined calcareous calculi which admittedly are formed by process of precipitation and find the fine structure of these, as seen by fracturing or crushing small portions, to be very similar to that of calcareous plates in arteries or thyreoid gland similarly treated.

All the evidence thus seems to point to the deposition of calcium salts in true ossification as being a secretory process of the osteoblast, while in areas of calcification it is a precipitation phenomenon.

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ANÆSTHESIA AND ANÆSTHETICS.

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By chance we have frequently discovered the typical drug of any group first. Only long search and much study have enabled us to find a better drug or one that is equally or more efficient and less toxic. Cocaine in spite of its many disadvantages, still holds its own, though it is being gradually replaced for most purposes by synthetic substitutes. Chloral, too, is still a highly esteemed hypnotic possessing advantages over most of its congeners. Amongst the anæsthetics the same holds true. Nitrous oxide was the first of the general anæsthetics to be successfully used. Discovered by Humphrey Davy in 1800, it was first used as a general anæsthetic by Horace Wells in 1844, but it did not come into favour as an anæsthetic until early in the twentieth century, as chemical manufacturers and instrument makers had not as yet solved the problems of cheap production and condensation into small tanks, nor a system of delivery to the patient. The second anæsthetic in order was ether. Its anæsthetic properties were discovered by Morton following the suggestion of the chemist Jackson and in 1846 it was first employed in surgery, producing perhaps one of the greatest revolutions which this branch of medicine has known. The third commonly used anæsthetic is chloroform whose anæsthetic action was discovered in 1847 by Fluorens and introduced by Simpson, of Edinburgh, as a general anæsthetic.

Let us briefly consider the advantages and disadvantages of these anæsthetics, as such a study will indicate that they all possess certain defects which suggest the need of further experimentation and the introduction of a more useful gas.

Chloroform.

Chloroform is a very potent anæsthetic. Air containing 2% of chloroform vapour rapidly produces anæsthesia with but a short period of stimulation and few reflexes. Reflex respiratory arrest is usually absent and little reflex salivation is produced either during induction or during the period of recovery. About 1% of vapour in air is sufficient to maintain surgical anæsthesia with complete absence of reflexes. Nausea during the period of recovery is much less than with ether. It is, however, a dangerous gas. Should the patient hold his breath in the early stages of induction, due either to a reflex or volition, high concentrations of chloroform may accumulate under the mask and the first inspiration after the arrest may contain concentrations which are toxic for the heart or the respiratory centre because the interval between the anæsthetic concentration and the toxic one is not great. Secondly in light chloroform anæsthesia the heart muscle is somewhat affected. Levy[1] has shown that when anæsthesia under chloroform is light, the heart of a cat may be rapidly thrown into fibrillation by the administration of adrenalin and many deaths during light chloroform anæsthesia are probably best explained on the basis of these observations. It is probable that adrenalin is secreted as a response to pain and asphyxia, as has been claimed by Cannon and others. Further, as cocaine, even in amounts that do not in themselves produce any symptoms, increases the sensitivity of the sympathetic endings in the heart and elsewhere to normal amounts of adrenalin, it is not remarkable that many cases of sudden cardiac death have occurred under light chloroform anæsthesia when cocaine has been employed as well for its local effect. There have been not a few cases of sudden death under light chloroform anæsthesia, for example tonsillectomy, where a very minor condition or surgical procedure has led to sudden death of a cardiac kind and I think this should as a rule be considered as having been due to cardiac fibrillation. These deaths do not seem to occur if anæsthesia with chloroform is deep.

Chloroform, unfortunately, shows its toxic action upon the heart not only during anæsthesia, but also subsequently. It leads to fatty changes which may especially in hearts that are not normal lead to death some hours or even days later. It also produces death after a delay of some days owing to its poisonous effects on the liver and this appears to occur especially in patients whose liver glycogen store is low.

Chloroform almost from the first depresses the centres in the medulla. The activity of the respiratory centre is decreased, the heart rate increased and the blood pressure falls almost from the earliest stage. This, too, is a serious factor in prolonged anæsthesia under chloroform.

Chloroform then is to be avoided, though it possesses one advantage which may lead to its employment in specific cases. It does not explode when mixed with air. However, both the thermocautery or the electric one may decompose the gas producing poisonous phosgene, but the risk from this source does not appear to be great.

Ether.

Ether is probably the most generally employed anæsthetic. Its stimulant phase is relatively long unless high concentrations of vapour are given. If these high concentrations are used, the reflex effect, namely, respiratory irregularity and increased salivary and bronchial secretion, are more pronounced. The latter reflex occurs in almost all

cases. This increased secretion can, of course, be prevented by the previous administration of atropine. This procedure is probably not used as frequently as it might be, largely due to a lack of a full comprehension of the action of atropine in the small doses (0.6 milligramme or $^1/_{100}$ grain) which would be employed. Six decimilligrammes *per os* (acting in fifteen or twenty minutes as a rule) or subcutaneously decreases nasal, buccal and bronchial secretion, but the heart rate is decreased, not increased, owing to a central stimulant effect of atropine and the fact that the dose given is not sufficient to depress the vagus endings in the heart appreciably. Respiration is if anything increased and blood pressure is unaffected. Spasm or high tonus in the gut or other smooth muscles is somewhat decreased, but the active movements of these organs are unaffected.

Ether affects the normal heart but little and a diseased heart very much less than chloroform. Only in ill-nourished infants have cases of post-operative death with fatty changes in the heart and liver been reported.

Ether stimulates the respiratory centre at first and respiration remains often within normal limits throughout the period of anæsthesia. In many cases heart rate increases but slightly and blood pressure remains normal or falls but slightly.

Its great disadvantage seems to be due to its relatively high solubility in water. This is twenty times as great as chloroform. Ether is therefore not so electively absorbed by the lipoids of the brain and proportionally more lodges in the rest of the body. Hence the prolonged period taken for its excretion and during this period ether produces both direct and reflex stimulation of the centres leading to nausea and vomiting. The relief of this condition deserves, I believe, further study and forms an argument for insisting on the anæsthetist sharing the responsibility for after treatment. There seems little doubt that the patients of certain anæsthetists suffer less from nausea than those of others. Whether this is due to the methods of administration or the use of such procedures as the prior administration of atropine or postoperative gastric lavage we do not as yet know.

Ether and air mixtures are explosive. Even as low a percentage of ether as 3% in air will explode. A mixture of nitrous oxide, oxygen and ether will also explode.

Both ether and chloroform produce other changes in the body not as yet fully understood. They both produce an immediate and marked rise in the hydrogen ion content of the blood stream and fall in the carbon dioxide combining power, which presently leads to an uncompensated alkali deficit.[2] They also cause an increase in the blood sugar and, as Stehle and Bourne[3] have shown, lead to an accumulation of phosphoric acid in the liver and to a post-operative increase in excretion of phosphates. Also acetone and β oxybutyric acid appear in the post anæsthetic period and this is probably an expression of the effect of these anæsthetics on the functions of the pancreas.

Nitrous Oxide.

Nitrous oxide is in some respects the best of these three anæsthetics. It has, of course, the disadvantage that the tanks containing it and the oxygen which must be employed if it is to be given for any period of time, are heavy and relatively expensive. The machines, too, which reduce the pressures of the gases and deliver them to the patient, are bulky and expensive. Yet the war has taught us that many patients, for example those suffering from shock, can be anæsthetized with relative safety with nitrous oxide, when ether or chloroform would be fatal. Its action is prompt. It produces practically no reflex effects and recovery is usually free from nausea. Late deaths from its use appear to be extremely rare if they occur at all.

Yet as a general anæsthetic it has one great disadvantage, namely that when given pure it produces asphyxia and hardly produces complete relaxation of the muscles. Even after the period of induction is over, the percentage of gas can scarcely be reduced beyond 93%, allowing only 7% of oxygen, hence oxygen want is always present. The blood of a patient anæsthetized with nitrous oxide is never saturated with oxygen and even if 15% of oxygen is given, the oxygen saturation of the blood falls from a normal of 92 to 90 in some fifteen to thirty minutes; indeed Greene states that if the blood is saturated to even 80% with oxygen no true anæsthesia is produced with nitrous oxide. It is probable that tissues under anæsthesia should be offered more rather than less oxygen than normal, because their ability to absorb oxygen is decreased. In consequence of these facts there are changes in the acid base equilibrium in the blood stream and probably in the tissues. At first there is a decrease in the hydrogen ion content, though the variations in carbon dioxide combining power are irregular. Later there is a fall in carbon dioxide combining power and a further increase in the hydrogen ion content. These changes are not as great nor as rapid as with ether or chloroform.[4] It is not remarkable that deaths occur especially in older persons or those whose hearts are enfeebled by disease, if they undergo a prolonged period of nitrous oxide anæsthesia.

Nitrous oxide and oxygen do not form an explosive mixture.

Ethylene.

The recognition of the disadvantages in these anæsthetics and also of the advantages shown by nitrous oxide led Dr. W. E. Brown to undertake some experimental work which resulted in the discovery in 1923[5] [6] of the anæsthetic properties of ethylene (C_2H_4). Carter and Luckhardt[7] had also been experimenting with this gas and published their results about the same time as Brown. As ethylene tanked in cylinders was readily available and could be administered with the machines already in use for nitrous oxide, it very soon was employed by numerous anæsthetists and doubtless in the past two years has been used by a hundred anæsthetists for many hundreds of cases.

Ethylene as an anæsthetic was discovered by Richardson in 1885 and mixtures of 85% to 87% of ethylene, sometimes rising to 90%, may be used to induce anæsthesia, while a concentration of 80% to 85% of ethylene with 15% to 20% of oxygen is sufficient to maintain surgical anæsthesia as a rule. Occasionally lesser amounts may be used. The induction period is short and appears to be free from stimulation or reflexes. The careful studies of Leake and Hertzman show that the effects of ethylene on the blood depend to a large extent on the amount of anoxæmia present. If no anoxæmia is present, as judged by the oxygen saturation of the blood, the hydrogen ion content rises slightly while the carbon dioxide combining power tends to fall, but both remain within normal limits even after an anæsthesia of forty minutes.[4] If anoxæmia is present, the change resembles that found in nitrous oxide. Recovery from ethylene is as rapid as under nitrous oxide and indeed without as much distress, owing to the presence of normal amounts of oxygen, but there is some postoperative vomiting, though large series of cases show that this is much less frequent than with ether. Ethylene has the disadvantage of possessing a somewhat unpleasant odour. This may in part be covered by the use of oil of cinnamon, but is unpleasant to the surgeon and others present. Its mixture with air is explosive, though not more so than a mixture of nitrous oxide, oxygen and ether and care must be taken that the cautery is not used. Postoperative deaths have not been reported. Complete relaxation in many patients can only be obtained with high concentrations which produce anoxæmia.

Ethyl Chloride.

Ethyl chloride is, of course, a dangerous anæsthetic and one that should not be employed for long periods of time. It probably possesses in a minor degree the disadvantages of chloroform and produces blood changes similar to that anæsthetic or ether.[2]

Acetylene.

Acetylene, introduced in 1923 by Gauss and Wieland[8] produces anæsthesia in a concentration of 65% to 75%. This allows an ample margin for oxygen. The hydrogen ion concentration of the blood appears to rise more markedly than with ethylene, though not to the same extent as with ether. It seems to be more variable as an anæsthetic and even more explosive than ethylene and has the same unpleasant odour.

It is evident that there is still the possibility of discovering an anæsthetic gas more potent than ethylene and having the advantages this gas shows in prompt recovery and little postoperative nausea.

Propylene.

As the anæsthetic properties of the alcohols rise disproportionately to their toxicity up to propyl or possibly butyl alcohol, there seemed reason for trying the higher members of the ethylene series. In consequence, Dr. Brown experimented with propylene and showed that it produced anæsthesia in cats in a concentration of about 40%. Anæsthesia may be maintained with concentrations varying from 31% to 25%, while concentrations of 65% were only slightly toxic. Recovery from this gas seems as prompt as with ethylene. There is no reflex stimulation and the gas seems to possess less odour than ethylene. It is, of course, equally explosive. In man anæsthesia is produced in a minute or two with concentrations of 50%, without unpleasant subjective symptoms.[9] Doubtless lower percentages would be successful, but have not as yet been tried. The wide margin that one has with this gas, should allow one to produce complete surgical relaxation without toxic effects. Its commercial production has been studied by Mr. L. J. Bonham[10] and it is as easy to prepare in a high degree of purity as ethylene.

Other Alcohols.

Preliminary experiments with the next highest member of the series, butylene, showed that it is an anæsthetic in even lower concentrations, but that there are indications of its having some peculiar toxic effects which are not as yet understood.

Methane appears to be a very weak anæsthetic requiring high concentrations which can only be obtained at more than normal pressures. It was apt to cause irregular respirations which gradually disappeared.

Propane produces anæsthesia in concentrations of approximately 90%, but there are extraordinary irregularities in its anæsthetic effects and the margin betwen toxic and anæsthetic concentrations appears to be very small. At present it does not appear that gases of this series are likely to prove to be satisfactory anæsthetic agents.

These studies appear to indicate that the unsaturated carbon series, especially that containing ethylene and propylene, are the best anæsthetic gases and of these two that propylene gives every promise of proving to be a highly satisfactory anæsthetic and better than anything that has been found up to this time.

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The Medical Journal of Australia

SATURDAY, JULY 18, 1925.

Interchange of Thought.

THIS issue of THE MEDICAL JOURNAL OF AUSTRALIA will be welcomed by our readers both on account of the value of the admirable articles that have been contributed by our Canadian colleagues and because a group of distinguished Canadian medical practitioners have manifested a kindly feeling toward their colleagues in Australia by sending them these articles. Last year Dr. N. D. Royle was asked to convey to a few selected practitioners in Canada and the United States of America an invitation to contribute articles to this journal. He carried the message faithfully when he visited North America to deliver with the late John Irvine Hunter the Doctor John B. Murphy Oration. Dr. F. N. G. Starr, of Toronto, accepted the invitation without hesitation and expressed his readiness to do even more than he had been requested to do. He suggested that he would approach some of the leading authorities in the various branches of medical science in Toronto and would collect articles that might be published in a special issue of this journal. In due course the articles were prepared, collected and dispatched. The proposal to devote an issue to them appeared to be so excellent that it was determined to carry it into effect. We desire to express our gratitude to Dr. F. N. G. Starr, Dr. D. E. Robertson, Dr. George S. Strathy, Dr. W. R. Campbell, Professor Duncan Graham, Dr. A. Almon Fletcher, Dr. Roscoe R. Graham, Professor James Crawford Watt and Professor Velyien E. Henderson for their valuable contributions and for the friendly spirit which induced them to send them. We are equally indebted to Professor Eric A. Linnell for a tenth article. Unfortunately the fact that this article has been published in *The University of Toronto Medical Journal* prevents us from making use of it.

It is a relatively rare privilege for medical practitioners to visit the old country and the other parts of the British Empire for the purpose of widening their outlook and of obtaining new points of view in connexion with medical matters. The training given to our students at the three medical schools in Australia is admirable, as is the teaching provided at the post-graduate courses. But a prophet is not always honoured in his own country and it has become almost a fashion to regard foreign doctrines as superior to our own. Although this mental attitude is untenable, it is always healthy and beneficial to contrast the doctrines of two schools of thought separated by space, by divergent methods and by customs. The Toronto School has a great reputation and its votaries can command attention in all parts of the world. The articles in this issue are worthy of close study and critical analysis. They should not be accepted because their authors are Canadians, but judged on their merits. If received in this way they will be found to be of special value to Australian practitioners, because they have been conceived in a scientific spirit and are dependent on originality of thought and fearlessness in treatment. Their didactic importance will be appreciated by all serious students.

Three articles on the treatment of *diabetes mellitus* will be read with avidity because some extraordinary work has been conducted in Toronto in regard to this disease and because of the fact that "Insulin" was discovered by workers in that University. The general practitioner may be gratified that all three of these articles are concerned with the treatment of diabetes, not with its pathology. Treatment, however, is far less important than ætiology, pathogenesis and pathology. Dr. Campbell holds the view that the extract of pancreas known as "Insulin" is identical with the hormone governing the metabolism of carbohydrate and employs the term suggested by Schäfer for this substance, namely insulin. The use of the same term for the ferment-like body normally produced within the body by the cells of the islands of Langerhans and for the therapeutic extract artificially prepared of pancreatic tissue tends to introduce some confusion. We have followed our usual practice of distinguishing a patented remedy by inverted commas and the use of an initial capital

letter; Schäfer's insulin carries no such differentiation. These remarks are made in explanation and not in criticism of these three admirable articles.

It is hoped that other articles or groups of articles from eminent teachers in universities outside Australia may be published in these columns in the future. They serve to bring the members of the medical profession in distant parts close together and to free science from the fettering limitations of insularity. It has been asserted over and over again that science knows no nation and that medicine is a great brotherhood. We shall have achieved part of our purpose if this departure from our usual routine will strengthen the bond of Empire and bring the medical profession in Canada and in Australia into more intimate contact.

THE NEW HOUSE OF THE BRITISH MEDICAL ASSOCIATION.

On July 13, 1925, Their Majesties the King and Queen opened the new House of the British Medical Association in Tavistock Square, Bloomsbury, London. The Archbishop of Canterbury performed the ceremony of dedicating the memorial gates. The proceedings were signalized by a dignity and pomp worthy of the occasion for the occupation of this handsome and stately building will be an epoch in the history of the Association.

Dr. W. N. Robertson, the delegate of the medical profession in Australia, headed the procession of representatives of the Branches in the outposts of Empire. It is fitting that Australia comes first, for in no other Dominion, Commonwealth or Colony has the British Medical Association developed as strongly or been as finely organized. The Australian Branches have achieved much. They comprise practically the whole of the practising medical profession and no medical practitioner who values his good name, can afford to stand outside. Dr. Robertson was presented to Their Majesties and was then given an opportunity to offer the President's Chair to the British Medical Association as a gift of the Branches in Australia. It will be remembered that the Federal Committee with the approval of all the Branches requested Dr. Alfred Cox, the Medical Secretary, to have a suitable chair made at its expense of Australian wood and to have attached to the chair a plate bearing an inscription of dedication. The chair has been designed by Sir Edwin Lutyens, the architect of the new House and the wood has been secured in London through the good offices of the High Commissioner and Mr. Joseph Davis. This handsome gift is an emblem of kinship and kindly feeling which will endure between the medical profession in the Commonwealth and that in the old country.

Current Comment.

CHONDROMA OF THE LUNG.

Benign tumours of the lung are of rare occurrence. Of the connective tissue series osteomata, chondromata and fibromata have been described and in recorded instances the tumours have as a rule been multiple and of small size. Although Virchow reported the occurrence of a true primary osteomata of the lung, it has been pointed out that some of the tumours so described have in reality been calcified foci resulting from tuberculosis or other diseases. The primary chondromata of the lung hitherto described have been small and have been regarded as arising from the bronchial cartilages.

Four instances of chondroma of the lung have recently been described by Dr. C. B. McGlumphy.[1] These tumours are of interest in view of the statement by Dr. McGlumphy that they did not arise by neoplastic proliferation of physiological elements, that is from cartilaginous structures in the wall of the bronchi. He claims that no analogous tumours have been described in the literature, with the possible exception of one recorded by Hart. All four tumours were of a lobulated, conglomerated type. In three of them the units were separated by a vascularized stroma containing abundant epithelial elements. Cellular elements of a somewhat similar nature were described by Hart in his case. Regressive metamorphosis was found in the centres of some of the masses. These areas gave the impression of some sort of hydropic change or even liquefaction. In several places the periphery of the tumours gave evidence of a gradual transition to connective tissue and the fibres of this tissue were so arranged that they finally formed a continuous mass resembling a true perichondrium. The interstitial areas appeared as thin tongue-like septa. They contained in addition to the epithelial structures a small amount of connective tissue and blood vessels. Sometimes the septa appeared to be formed mainly by the epithelial structures. These struc-

[1] *The Journal of Cancer Research*, December, 1924.

tures appeared chiefly as longitudinal axial sections of ducts with a lumen of more or less constant width lined by epithelium on both sides. Cross sections of the ducts were not seen and it is concluded that the structures were not ducts, but flat sacs or pockets with complicated ramifications. The tumours are described in short as a conglomeration of closely packed chondromatous bodies the surface of each of which was covered by a thin epithelial layer. In places separation of the tumour from the surrounding lung tissue was maintained by the epithelial structures. The epithelium was cylindrical, but in the centre of the tumour what is regarded as its essential element was seen in the form of ciliated epithelium. No transition could be found between this epithelium and the epithelial structure of the lung.

In the fourth tumour described by Dr. McGlumphy the epithelial layers between the cartilaginous units were lacking. The common features of all four, however, were so striking that Dr. McGlumphy raises the question as to whether the fourth tumour was not of the same nature as the other three. He suggests that it had lost its epithelial component in the course of its development. Three of the four tumours were situated in the lower lobe of the left lung; in one instance the site was not recorded. Two of the tumours were found near and below the hilum. The largest was two centimetres in diameter. As stated before Dr. McGlumphy points out that the tumours had no connexion with any physiological tissues, but gave the impression that they had arisen from aberrant structures. The uniformity of localization in his opinion made it possible that the material forming the tumours corresponded to a structure which had disappeared in phylogenesis. From the standpoint of human anatomy such a structure would probably have to be classified as a supernumerary bronchus. He suggests that the nature and origin of this bronchus should be investigated by a study of the comparative anatomy of the lung in lower animals. In view of the two important components of the tumour he considers that the name adenochondroma is justified.

LYMPHOBLASTIC ERYTHRODERMIA.

THE term erythrodermia was first used by Besnier to describe reddened areas of the skin such as are seen in one of the stages of *mycosis fungoides*, in *pityriasis rubra pilaris* and some other conditions. This red appearance is usually accompanied by some degree of infiltration. Like so many other words used in medical literature its use has been extended and it has been applied to almost any red patch of the skin of uncertain nature. In *mycosis fungoides* the red appearance of the skin is accompanied by the tumour formation characteristic of the disease and in *pityriasis rubra* scaling takes place. In 1921 Dr. J. H. Sequeira and Dr. P. N. Panton described three cases in which erythrodermia was accompanied by a specific change in the blood. They have recently had an opportunity of studying another case of the same nature in which an autopsy was obtained, and have made an interesting report of their findings.[1] In all they give the history of five patients. It is not necessary to describe the condition of these patients in detail. Four were males and their ages varied from twenty-three to sixty-four. Generalized redness of the skin was present. It may begin as an eruption of striated and retiform character, but ultimately the eruption becomes universal. The skin retains its peculiar red colour for years, the tint varies from day to day, but a peculiar, dull, pinkish-red is recognizable. Slight infiltration of the skin is present with some scaling and pruritus is a prominent feature. In one of the four instances the spleen was considerably enlarged. Glandular enlargement was present, but this was not very pronounced. Some degree of anæmia of a secondary type is usually present and may be severe. The characteristic changes were found by Dr. Sequeira and Dr. Panton in the white cells. A relative and absolute increase of the lymphocytes was found and the small lymphocytes were mainly affected. The total number of leucocytes varied from 8,000 to 60,000 per cubic millimetre and of these the small lymphocytes sometimes formed as much as 80%. Dr. Sequeira and Dr. Panton point out that this blood picture occupies a place between that of secondary anæmia and that of chronic lymphatic leuchæmia. In the latter condition a considerably higher count and a greater relative proportion of lymphocytes are found. In a series of sixteen cases of chronic lymphatic leuchæmia the total white cells averaged 96,300 and the lymphocytes 79.8%. In the five cases of lymphoblastic erythrodermia recorded by these observers the numbers were respectively 22,300 and 63.7%.

The important points to be considered in connexion with this report are the pathogenesis and the relationship of the condition to other diseases. The pathogenesis is not actually considered by Drs. Sequeira and Panton. They hold that if the syndrome erythrodermia with lymphæmia is sought, other cases will be discovered. Further research must be carried out before much will be known of the pathogenesis. When this has been determined, the relationship of the syndrome to other conditions will possibly become clear. Dr. Sequeira and Dr. Panton throw doubt on the existence of this relationship. They recognize a state of erythrodermia preceding the tumour stage of *mycosis fungoides* which is unaccompanied by changes in the blood, and they have occasionally seen patients suffering from leuchæmia whose skin manifested no signs of local infiltration. In the five cases described by these authors no tumour formation occurred. Furthermore the blood picture bore no resemblance to that of myeloid leuchæmia in which infiltration of the skin is sometimes found. They point out that although the blood picture approached that of lymphatic leuchæmia, at no stage was it in any sense typical of that condition. The erythrodermia developed independently of the lymphæmia.

[1] *The Quarterly Journal of Medicine*, April, 1925.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

The Effect of Severe Exertion on Athletes.

W. PARRISIUS (*Münchener Medizinische Wochenschrift*, November 14, 1924) examined one hundred competitors in the German ski championship before and after the contest. Only twenty-four were found to be perfectly sound, whilst the number with thyreoid enlargement was very high. Many had functional cardiac disturbances and the actual winner had an organic heart lesion. The blood pressure was increased before the race probably owing to psychical causes. In eighty-four cases it was lowered after the contest. The pulse rate when observed afterwards was much accelerated and in many cases it was irregular and unequal. The average rectal temperature was 39.5° C. The hæmoglobin content of the blood was increased 5% to 30% above normal.

Composition of Glomerular Urine.

J. T. WEARN AND A. N. RICHARDS (*American Journal of Physiology*, December, 1924) have devised a method for obtaining fluid from the glomeruli of frog's kidneys and by its use have obtained some comparisons between the composition of glomerular urine and bladder urine. The essential part of the apparatus used for drawing fluid from Bowman's capsule is a sharp-pointed capillary pipette made of quartz tubing. Qualitative analysis of the fluid withdrawn from the capsule was made in small capillary tubes. When the blood flow through the glomerular capillaries was rapid, neither the glomerular fluid nor the bladder urine contained protein. In two experiments no reaction for sugar could be obtained with Benedict's qualitative solution with either glomerular or bladder urine. In these cases examination of the blood revealed the absence of sugar. In other cases sugar was injected into the blood. In all cases in which the blood sugar level was 0.05% or lower, glomerular urine reduced Benedict's solution; bladder urine did not. In the single experiment in which the blood sugar was found to be higher than 0.05%, both fluids reduced Benedict's solution, glomerular urine more intensely than bladder urine. Glomerular fluid of fasting frogs yielded a heavy precipitate in the test for chlorides, while the bladder urine gave only slight turbidity. When the frogs were kept in distilled water, the bladder urine was free from chlorides, while the glomerular fluid contained chlorides. The glomerular fluid contained urea, so that urea is secreted by the glomeruli. The dyes indigo-carmine, phenolsulphonephthalein and methylene blue are eliminated from the frog's circulation by way of the glomerulus. The evidence obtained favours the view that substances are filtered off in the glomerulus and that certain of them are subsequently reabsorbed in the tubules.

The Maximum of Human Power and its Fuel.

THE maximum physical power of which a man in the prime of his strength and at the height of athletic training is capable, is a matter with many important bearings. It is probable that the rowing of a crew in a racing boat with sliding seats is a form of exercise in which a greater total energy expenditure is attainable for periods of five to twenty minutes than under any other conditions. Yandel Henderson and H. W. Haggard (*American Journal of Physiology*, April, 1925) have studied the record-breaking Yale University 1924 crew which led its speediest rivals in the last Olympic championship by five boat lengths at the end of a course of two thousand metres. The energy expended in rowing an eight-oared racing boat for the various distances and speeds occurring in contests has been determined by three different methods. The results of the diverse methods agreed satisfactorily. They indicated that the maximum power exerted was from 0.45 to 0.57 horse power per man or expressed in heat equivalents, 4.8 to 6.0 calories per minute, with a total energy expenditure of nineteen to thirty calories per minute, or thirteen to twenty times the basal rate. The smaller figure is for twenty-two minutes during a four mile race, while the higher figures are applicable to the more intense exertion and greater speed for about six minutes in races of two thousand metres. The corresponding figures for the volume of oxygen consumed per minute are three and a half to four litres. The oarsman thus incurs oxygen deficits of four to eight litres or more. The most significant result of these observations is the conclusive evidence which they afford, that in whatever proportion fat and sugar are being burned during rest just before the exercise, they are burned in nearly the same proportion to produce the energy for doing work or for the recovery process in the muscles. A man can make an intense exertion, although rather disadvantageously, on a combustion almost entirely of fat from his own body. There is great advantage in increasing the proportion of sugar in the diet even to the extent of giving a hundred grammes of some simple sweet a half to three-quarters of an hour before any prolonged contest.

Culture Macrophages, Epitheloid Cells and Giant Cells.

W. H. LEWIS and M. R. LEWIS (*American Journal of Physiology*, March, 1925) have demonstrated that in hanging drop cultures of pure blood the monocytes undergo considerable changes. Some of them exhibit high phagocytic activity especially for red blood corpuscles. Such cells become indistinguishable from the large macrophages so abundant in the tissue spaces, spleen and liver. Other monocytes, not so favourably located for contact with the red cells, appear to absorb ultramicroscopic material and fat globules accumulate about the central area. These cells are precisely like the epitheloid cells from the tubercles in smear preparations from fresh tuberculous lungs. Some of the epitheloid cells in the hanging drop hypertrophy and form multinucleated giant cells. Many intermediate forms are also seen. The monocytes, macrophages and epitheloid cells thus appear to be merely different functional states of the same cell type. Thus the macrophages are probably derived from the monocytes of the blood, as are also the epitheloid cells so characteristic of tuberculous lesions.

Kidney Size and Diet.

T. B. OSBORNE, L. B. MENDEL, E. A. PARK and M. C. WINTERNITZ (*American Journal of Physiology*, March, 1925) have studied the effect of diets rich in protein on the size of the kidney. With rats on high protein diets the kidneys become greatly enlarged, the increments in size often exceeding 50% of the normal weight of these organs. Histological examinations have failed to disclose changes of an inflammatory or degenerative nature. The renal enlargement has occurred without enlargement of the heart. Comparable changes in kidney size can be brought about by the inclusion of large quantities of urea in the diet. The excretory functions of the kidney have been greatly augmented by the administration of large quantities of a variety of inorganic salts without, however, bringing about the hypertrophy that developes through the necessity of eliminating the nitrogenous waste. The rapidity with which the kidneys respond by enlargement to large increments of protein in the diet is surprising; in many instances distinct changes have resulted within a week. When the ration is changed to a low protein level after a regimen of high protein a retrogression in the size of the kidney ensues.

A Parathyreoid Hormone.

J. B. COLLIP (*American Journal of Physiology*, March, 1925) has prepared an extract which he believes represents in potent form and a fair degree of purity the essential hormone of the parathyreoid gland. It has been found that tetany in parathyreoidectomized dogs can be either prevented or controlled by the administration of this extract by subcutaneous injection. It has been shown that the administration of the extract to normal dogs is followed by a rise in the level of blood calcium. By successive injections of the extract at intervals of twenty minutes to eight hours a condition of profound hypercalcæmia has been produced. Typical symptoms are manifested by dogs in a condition of extreme hypercalcæmia and death

may ensue. Besides increase in calcium there are also other profound changes in the mineral composition of the blood and in other blood constituents. The extract has been used in a case of infantile tetany and a remarkable result was obtained. It has also been used in other clinical conditions such as nephritis with œdema and the vomiting of pregnancy. No claim for the clinical effectiveness of the extract in conditions other than those with definite hypofunctions of the parathyreoid gland, is made at this juncture. The results so far obtained are suggestive only.

BIOLOGICAL CHEMISTRY.

Radiation of Rickets-Producing Ration.

H. STEENBOCK and M. T. NELSON have brought forward evidence dependent upon histological methods to show that a ration which induced rickets in rats, can be made definitely antirhachitic by the simple expedient of exposing it to ultra-violet light (*Journal of Biological Chemistry*, November, 1924). The authors have fed rats upon a ration composed of wheat, corn, gelatine, wheat gluten, sodium chloride and calcium carbonate. The corn is yellow corn. The authors state that one of the difficulties in producing rickets in rats is the prevention of ophthalmia and of infections of the respiratory tract sufficiently long to allow complete exhaustion of the reserves of antirhachitic factor to take place. With yellow instead of white corn insufficiency of vitamin *A* can be long postponed. Sooner or later the rats gave evidence of rickets as indicated by a peculiar flat-footed shambling gait, with a disinclination to "sidestep" as the rat usually does when suddenly disturbed. Oftentimes the rats were observed to lie comfortably curled up on their backs, feet in the air in an apparent attempt to relieve the limbs of the pressure of the body weight. Invariably the animals were plump and well behaved. The ration was exposed at a distance of sixty centimetres to the light of a quartz mercury vapour lamp giving off ultra-violet and other rays for a period of thirty minutes. The ration in quantities of ten grammes was spread in a thin layer. Data were obtained from rats that were fed on the ration until they developed severe rickets. The ration was then irradiated and the animals fed on the irradiated ration from three to nineteen days. Histological examination of radii and ulnæ showed that calcium deposition had taken place. Sections stained with silver nitrate showed that no deposition of lime had occurred after three days' feeding with irradiated ration, that a narrow line of calcium deposit was present after seven days on the irradiated ration, that the line materially widened after feeding ten days and that almost complete calcification had taken place at the end of twenty-one days. Except for a narrow fringe proximal to the epiphysis the deposits of lime represent permanent deposits with definitely formed trabeculæ. Suitable controls were used on animals that were fed with various amounts of food, on animals with a ration supplemented by cod liver oil and on irradiated animals. The authors consider that the absence of radiant energy explains the failure of assimilation of lime in the mature goat, in the growing chick and in the pig previously observed by them.

Organic Phosphorus in Urine.

G. E. YOUNGBURG and G. W. PUCHER have considered the analytical methods suitable for the estimation of organic phosphorus in urine (*Journal of Biological Chemistry*, November, 1924). These methods fall into two groups: (i.) those in which the total and the inorganic phosphorus are each separately determined, the difference representing the organic fraction and (ii.) those in which the inorganic phosphates are removed by precipitation and the organic phosphorus determined separately in the filtrate. The authors consider it doubtful whether satisfactory estimations can be made by the first method. They have therefore turned their attention to the accurate removal of inorganic phosphates and subsequent determination of the residual phosphorus. As precipitants for inorganic phosphates they have tested barium hydroxide, barium chloride, calcium chloride, ferric chloride, magnesium citrate and magnesia mixture. They have chosen the removal of inorganic phosphates with magnesia mixture and the subsequent determination of organic phosphorus in the filtrate by a modification of the colorimetric method of Bell and Doisy. They believe that their method gives somewhat low results owing to the volatilization of phosphorus when heating with sulphuric and nitric acids to destroy organic matter. The authors have found much fluctuation in the figures obtained in normal men for the excretion of organic phosphorus both during the day and at night. A change of 100% in successive two hourly periods has been frequently observed and even larger changes may occur from time to time. The authors have found it impossible to predict the excretion of any single individual. The authors consider that the output of organic phosphorus per kilogram of body weight is not constant. In twelve normal persons the lowest value for twenty-four hours was 0.089 milligramme and the highest value 0.187 milligramme with an average of 0.131 milligramme per kilogram of body weight. The figures given by the authors are much lower than those found by others except those of Mandel and Oertel. They have also not observed the relation to volume of the urine which has been stated by others to exist. Some data are also given of results in old people, children and persons suffering from diabetes, syphilis, tuberculosis and arteriosclerosis and in persons recovering from fractures and laparotomy.

Diastase in Cerebro-Spinal Fluid.

ISAAC COHEN (*Biochemical Journal*, February, 1925) has made a study of the occurrence of diastase in human cerebro-spinal fluid. He has used for the detection of diastase a modification of the method of Dodds in which cerebro-spinal fluid is mixed with a solution of phosphates to serve as a buffer and the mixture added to a standard dextrin solution. The substances are kept in contact at 37° C. for half an hour. The amount of change is determined by comparing the colour of a part of the treated dextrin solution when treated with iodine with that of the standard similarly treated. The author estimated the amount of diastase in the cerebro-spinal fluid of twenty-eight persons suffering from various affections as tabes, syphilis, uræmia, tuberculous meningitis and neuritis and also in healthy persons taken during life. In all except two not a trace of diastatic enzyme was found. In two traces of diastase could be detected in the cerebro-spinal fluid removed at autopsy. Diastase was found present in 20% of the cerebro-spinal fluids removed seven hours after death. The author regards this appearance of diastase as a *post mortem* effect occurring about seven hours after death. The enzyme was not found in fluid removed less than seven hours after death.

Glutathione in Tissues.

H. E. TUNNICLIFFE describes the occurrence and quantitative estimation of glutathione in various tissues (*Biochemical Journal*, January, 1925). He finds that glutathione occurs in the tissues of normal animals chiefly in the reduced form with sulphhydryl groups. The sulphur equivalent of the sulphhydryl groups of the glutathione present accounts for the greater part of the soluble "neutral sulphur" present in the tissues. The method of estimation has been to extract a known weight of tissue with water containing trichloracetic acid. The clear extracts which are free from protein are titrated with $^{N}/_{100}$ iodine solution, sodium nitroprusside being used as an external indicator. Various controls have shown this method to be satisfactory. Estimations have also been made of the sulphate sulphur and total sulphur in tissue extracts. The difference gives soluble organic sulphur. This figure corresponds well with the value obtained for sulphur present as SH groups. Figures are given for the quantities found in various tissues. The possibilities of interference between the — SH and the — SS — forms of dipeptide were considered and care was taken to diminish the action of factors likely to disturb the equilibrium.

Medical Societies.

THE ALFRED HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE ALFRED HOSPITAL CLINICAL SOCIETY was held on March 31, 1925, at the Alfred Hospital, Prahran, Victoria, MR. FAY MACLURE, O.B.E., the President, in the chair.

Foreign Body in the Chest.

DR. F. E. JONES reported the case of a woman who had come into hospital in 1911 with symptoms suggestive of chronic appendicitis. A skiagram of the kidney and ureter had revealed no abnormality. The appendix had been removed, but the symptoms persisted. Further questioning had elicited the following history. In the year 1890 she had fallen whilst carrying a bottle of beer and cut herself near the right costal margin. She had been very ill for some time. A scar which had previously passed unnoticed, being covered by a pendulous breast, was found.

A second skiagram higher than the first had revealed a large opaque foreign body lying across the tenth and eleventh ribs. At a subsequent operation a large piece of bottle glass had been removed from a cystic cavity with fibrous walls filled with dark fluid. The woman had made an uninterrupted recovery.

Carcinomata of the Mouth.

DR. J. LOVE showed a male, aged sixty-eight years, who had presented himself in April, 1924, complaining of a warty growth in the floor of the mouth near the frenum of the tongue. This had been excised and the raw surface deeply cauterized with the diathermy needle. The pathologist had reported the growth to be a papilloma showing carcinomatous change. Ten months later the man had returned on account of a small ulcer on the margin of the tongue, opposite the last molar tooth on the right side. This had been excised and treated with diathermy. The pathologist had reported the growth to be a squamous-celled carcinoma. Dr. Love remarked on the unusual occurrence of two distinct carcinomata in the one mouth. Both foci appeared to be perfectly healed and no abnormality was discoverable in the glands.

MR. A. J. TRINCA said that papillomata in any situation showed a definite tendency towards malignancy and should therefore be extirpated. Diathermy was an excellent agent for this purpose. In cancer of the tongue he thought that the cervical glands should not be attacked by operation.

MR. HAMILTON RUSSELL agreed in the main with Mr. Trinca. He recalled the case of a man who was alive and well fifteen years after operation for cancer of the tongue. He quoted a passage from the first edition of Cheyne and Burghard's book in which it was advised to remove the local condition only and not to touch the glands, unless they were observed at a later date to enlarge. This passage had been removed from later editions, but Mr. Russell believed that the advice therein still held.

Fracture of the Neck of the Femur.

DR. M. ASHKENASY reported the case of a male, aged thirty-six years, who for the previous eighteen months had complained of swellings and pain in many joints, most particularly in the left hip joint. The condition had been regarded as one of rheumatoid arthritis and treated as such, with the result that the symptoms largely disappeared, excepting that the left hip joint showed no improvement. Further examination of this joint had demonstrated wasting of the thigh and shortening of the leg, A skiagram had disclosed to the astonishment of the observers, an intracapsular fracture of the neck of the femur.

Dr. Ashkenasy said that there was no history of injury. The radiogram revealed no signs of osteoarthritis. It was remarkable that the man still walked on the leg without support.

DR. C. ADEY, discussing the skiagram, pointed out that there were no signs of osteoarthritis and no rarefaction of the bones. The clean cut fracture surface of the upper fragment suggested the possible presence of a myeloid sarcoma.

MR. HAMILTON RUSSELL, discussing ununited fractures of the femoral neck, said that the treatment was very difficult. Bone pegs were not successful as a rule. He advised removal of the head fragment, placing of the upper end of the lower fragment in the acetabulum and subtrochanteric osteotomy of the femoral shaft which was then abducted. The results were excellent, a strong, stable limb resulting.

DR. J. F. MACKEDDIE said that rarefaction of bones in cases of osteoarthritis was rare.

Compression Paraplegia.

DR. J. F. MACKEDDIE reported the case of a male, aged fifty-three, suffering from a compression paraplegia. Deformity of the dorsal spine with gibbosity was present. One leg was in a state of flaccid paralysis, the other spastic. Lumbar puncture had been performed and the fluid obtained had been found to be rich in protein. Fluid from a *cisterna magna* puncture was normal. "Lipiodol" injected was seen in the skiagram to be arrested in the region of the gibbosity. There was a large amount of new bone apparent in this region in the skiagram.

Dr. Mackeddie thought that operation was indicated for the relief of the compression.

Myositis Ossificans Traumatica.

MR. HUGH TRUMBLE, M.C., showed a boy, aged seventeen years, who six weeks before had been struck on the arm by a cricket ball. Two days later a firm swelling had appeared at the site of injury just above and anterior to the medial epicondyle. This had been regarded as a tense hæmatoma. A skiagram taken at the time showed no bony injury. On examination the lump appeared to have diminished in size, but was bony hard to the touch and moved over the humerus with a fine crepitus. A skiagram of recent date showed an oval shadow apparently caused by a delicate bony structure. The case was regarded as one of *myositis ossificans traumatica.*

Subacute Bacterial Endocarditis.

DR. F. K. NORRIS read the clinical history of a female patient, aged seven years, who had been admitted to the Alfred Hospital on December 26, 1924, with pain in the chest and constipation of one day's duration. On examination the child had been flushed and cyanotic and a systolic thrill had been present over the base of the heart with a harsh systolic murmur all over the heart, apparently maximal about the mitral area.

The child had been treated at Dr. Norris's Out-patient Clinic before this for cardiac trouble associated with cyanosis and Dr. Norris had regarded her cardiac condition as congenital, possibly involving the septal area. The temperature at admission had been 37.8° C. (100° F.) with pulse rate of 112 and from then on till March 17, 1925, when she was discharged, the temperature had run a swinging course, being up to 40° C. (104° F.) at times. Blood culture on January 11, 1925, had resulted in the growth of a moderately long chained hæmolytic streptococcus. The test had been repeated at intervals. On January 18, 1925, streptococci had been grown, on February 20, 1925, no growth had resulted and on March 7, 1925, streptococci had again been grown. About fourteen days after admission a sudden right pleural pain had developed, but this had soon disappeared; it had apparently been embolic in origin. This had been the only sign of emboli while the child was under observation, examination of the retinæ and urine failing to reveal any signs of microscopical emboli.

On January 21, 1925, the erythrocytes had numbered 5,800,000 per cubic millimetre and the white cells 30,000 and on January 29, 1925, the erythrocytes had numbered 5,240,000 per cubic millimetre and the white cells 22,600. The hæmoglobin value had been 100%. On February 5,

1925, sixty-five cubic centimetres of antistreptococcal serum had been given subcutaneously and intravenously, but with no improvement.

Except for a week in the middle of February the child had been under observation from December 26, 1924, till March 17, 1925; during this period the systolic murmur had become gradually harsher, though there were times when it was soft and blowing and its place of localization, the mitral area, was more easily demonstrated. There had been no enlargement of the heart and with rest in bed the child had been perfectly comfortable. In the week referred to above the mother had had the child at home, but would not keep her in bed, hence the return to hospital with dyspnœa and cyanosis. The child had ultimately been discharged on March 18, 1925, to the Austin Hospital. At no time had the spleen been palpable nor had any petechiæ been present.

MR. A. J. TRINCA referred to improved methods in obtaining blood cultures due to accuracy in the determination of the alkalinity of cultures in terms of the hydrogen ion concentration.

DR. J. F. MACKEDDIE remarked that in this case there was apparently no evidence of a previously damaged valve and so possibly more hope for treatment. He questioned the possibility of a primary pyæmic condition. Horder had recommended alternating an autogenous vaccine and an autogenous serum.

British Medical Association News.

SCIENTIFIC.

Corrigenda.

DR. A. P. DERHAM informs us that his remarks in the discussion on the treatment of pertussis at a meeting of the Melbourne Pædiatric Society held on November 11, 1924, have been incorrectly reported in the issue of July 4, 1925 (page 24). He states that he considers afebrile rather than febrile bronchitis an important prodromal sign of pertussis and that with the use of moderately large repeated doses of pertussis vaccine (Commonwealth Serum Laboratories) in about fifty primary and secondary contacts of a patient with pertussis all under one year of age only twenty babies developed pertussis. With two or three exceptions these ran a remarkably mild and a comparatively short course. Children not treated prophylactically ran the usual course. He considers that these figures at least suggest that the prophylactic use of pertussis vaccine is of some value.

"SPIRITUAL HEALING."

WE have been asked by the Honorary Secretary of the New South Wales Branch of the British Medical Association to publish the following letter and *questionnaire* which have been received by him from the Chairman of the Medical Research Group of the British Medical Association. We understand that similar letters have been addressed to the Honorary Secretaries of the other Branches of the Association in Australia.

Medical practitioners in all States having information concerning the persons who attended Mr. Hickson's meetings for the purpose of treatment of ailments, are requested to send this information either to Dr. E. Rowland Fothergill or to the Honorary Secretary of the Branch to which they are attached.

This invitation is addressed particularly to those practitioners whose patients were treated by Mr. Hickson, as in these cases the diagnosis has been made by those competent to recognize disease. The evidence of the doctors who attended the meetings without personal knowledge of the conditions from which the patients were suffering, is actually of less scientific value.

Probably you have read in the medical press that last July there was formed a medical research group to inquire into "Spiritual Healing."

The group is formed of Sir Robert Armstrong-Jones, Dr. Helen Boyle, Dr. H. C. Bristowe, Dr. William Brown, Dr. Charles Buttar, Mr. W. McAdam Eccles, Dr. Letitia Fairfield, Dr. E. R. Fothergill (Chairman), Dr. J. G. Porter-Phillips, Dr. Mary Scharlieb, Sir J. Purves-Stewart, Mr. E. B. Turner, Dr. Jane Walker, Dr. Stanley Bousfield (Honorary Secretary, 10 Albion Street, W. 2).

The Anglican and Roman Churches as well as the free churches have appointed representatives to meet us and already several conferences have been held.

The missions held by Mr. Hickson in various parts of the world have been noticed by the group and his recent book "Heal the Sick" referring to these missions has been read by us.

It has been decided therefore to make inquiries of medical practitioners in several of the districts where these missions have been held in order to provide our group with first hand medical information.

Your assistance would be much valued to this end and in order to make it as easy as possible for you a short *questionnaire* is enclosed. Any further information you can give as well as copies of any papers, circulars *et cetera* you may have by you would be of the greatest assistance to us.

Could you please issue to suitable doctors a copy of the *questionnaire* with any explanation you care to add and ask them to send their replies and all enclosures either direct to me or, if you prefer it, through yourself. Would you please ask for replies to be posted not later than two weeks after receipt of your letter.

Yours faithfully,

(Signed) E. ROWLAND FOTHERGILL.

16, Brunswick Place,
Hove, Essex, England,
May 6, 1925.

Questionnaire.

1. Was any systematic medical examination with diagnosis made of patients before they were seen by Mr. Hickson?
2. Were these diagnoses confirmed (*a*) by a second medical opinion and/or (*b*) by microscopical examination in suitable cases?
3. Were any of these patients claimed to have been cured?
4. Was any medical examination made shortly afterwards of such patients? If so, with what results?
5. Were these examinations confirmed (*a*) by a second medical opinion and/or (*b*) by a microscopical examination in suitable cases?
6. Have any of the patients claimed to have been cured been medically reexamined after some considerable time? If so, with what results?
7. Was the second medical opinion referred to in questions 2 and 5, given by recognized specialists?
8. (*a*) Do you consider that any of these cures can be accounted for as being due to means ordinarily adopted by doctors?
   (*b*) If so, what were the means used?
   (*c*) If not, how do you account for the cures affected?
9. About what was the percentage of patients claimed to have been cured out of those seen by Mr. Hickson?
10. Have the Hickson missions to your knowledge had any permanent or temporary influence for good or evil on the mental and/or physical health of those amongst whom the patients lived? If so, in what way?

(In replying to questions it would be of great value if you could add the diagnosis.)

NOTICE.

Owing to the fact that Thursday, July 23, 1925, has been proclaimed a public holiday in New South Wales in honour of the arrival of the visiting portion of the American Fleet, the issue of The Medical Journal of Australia of July 25, 1925, will be posted on Friday, July 24, one day later in the week than usual.

Books Received.

NEWER METHODS OF OPHTHALMIC PLASTIC SURGERY. By Edmund B. Spaeth, M.D., F.A.C.S. 1925. Philadelphia: P. Blakiston's Son & Company. Royal 8vo., pp. xix. + 258 with 168 illustrations. Price: $5 net.

SURGICAL TREATMENT OF PULMONARY AND PLEURAL TUBERCULOSIS, by J. Gravesen, M.D. (Copenhagen), with a foreword by S. Vere Pearson, M.D., M.R.C.P. 1925. London: John Bale, Sons & Danielsson, Limited. Demy 8vo., pp. xii. + 155 with 87 illustrations. Price: 10s. 6d. net.

BULLETIN NUMBER XI. OF THE INTERNATIONAL ASSOCIATION OF MEDICAL MUSEUMS AND JOURNAL OF TECHNICAL METHODS, Editorial Committee Maude E. Abbott, Major James F. Coupal. 1925. New York: Paul B. Hoeber, Incorporated. Demy 8vo., pp. xii. + 151. Price: $3.

AN INTRODUCTION TO SEXUAL PHYSIOLOGY FOR BIOLOGICAL, MEDICAL AND AGRICULTURAL STUDENTS, by F. H. A. Marshall, F.R.S. 1925. London: Longman's, Green & Company. Demy 8vo., pp. xii. + 167.

THE CEREBRO-SPINAL FLUID IN CLINICAL DIAGNOSIS, by Godwin Greenfield, M.D., B.Sc., M.R.C.P. and E. Arnold Carmichael, M.B., Ch.B. 1925. London: Macmillan & Company Limited. Demy 8vo., pp. x. + 272. Price: 12s. net.

THE COMING OF BABY, by Lucy E. Ashby and Kate Atherton L. Earp, with a foreword by Sir James Cantlie, K.B.E., M.A., M.B., F.R.C.S., V.D. 1925. London: The Scientific Press Limited. Foolscap 8vo., pp. 92. Price: 2s. net.

SEX AND EXERCISE, A STUDY OF THE SEX FUNCTION IN WOMEN AND ITS RELATION TO EXERCISE, by Ettie A. Rout, foreword by A. C. Haddon, M.A., Sc.D., F.R.S. 1925. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. vii. + 97.

Medical Appointments.

Dr. Owen Meredith Moulden (B.M.A.) has been appointed Honorary Gynæcologist to the Mental Hospital, Parkside, South Australia.

* * * *

Dr. Eugene Abraham Matison (B.M.A.) has been appointed Honorary Surgeon for Ear, Nose and Throat to the Mental Hospital, Parkside, South Australia.

* * * *

Dr. John James O'Grady has been appointed Honorary Ophthalmologist to the Mental Hospital, Parkside, South Australia.

* * * *

Dr. D. M. Steele (B.M.A.) has been appointed Quarantine Officer, Port Lincoln, South Australia.

* * * *

Dr. C. O. F. Rieger (B.M.A.) has been appointed Deputy Quarantine Officer, Port Lincoln, South Australia.

* * * *

Dr. William Henry Golding has been appointed Government Medical Officer at Home Hill, Queensland.

* * * *

Dr. Harold Henry Field-Martell (B.M.A.) has been appointed a Justice of the Peace for the Fremantle Magisterial District, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

Department of Public Health, Tasmania: Director.

St. Vincent's Hospital, Sydney: Relieving Honorary Radiologist.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
South Australian: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

July 21.—Tasmanian Branch, B.M.A.: Council.
July 22.—Victorian Branch, B.M.A.: Council.
July 24.—Queensland Branch, B.M.A.: Council.
July 24.—Eastern Suburbs Medical Association, New South Wales.
July 28.—New South Wales Branch, B.M.A.: Medical Politics Committee: Organization and Science Committee.
July 30.—New South Wales Branch, B.M.A.: Branch.
July 30.—South Australian Branch, B.M.A.: Branch.
Aug. 4.—Tasmanian Branch, B.M.A.: Council.
Aug. 5.—Victorian Branch, B.M.A.: Branch.
Aug. 6.—Section of Orthopædics, New South Wales Branch, B.M.A..
Aug. 7.—Queensland Branch, B.M.A.: Branch.
Aug. 11.—Tasmanian Branch, B.M.A.: Branch.
Aug. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
Aug. 13.—Victorian Branch, B.M.A.: Council.
Aug. 13.—South Australian Branch, B.M.A.: Council.
Aug. 13.—New South Wales Branch, B.M.A.: Clinical Meeting.
Aug. 14.—Western Australian Branch, B.M.A.: Council.
Aug. 14.—Queensland Branch, B.M.A.: Council.
Aug. 18.—Tasmanian Branch, B.M.A.: Council.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor," The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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